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GLASGOW HOSPITAL REPORTS

EDITED FOR THE COMMITTEE

BY

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VOL. III.

WITH FIFTY-THREE ILLUSTRATIONS

GLASGOW
JAMES MACLEHOSE AND SONS

Publishers to the University

1901

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THE ANATOMICAL AND PATHOLOGICAL PREPARATIONS OF DR. WILLIAM HUNTER IN THE HUNTERIAN MUSEUM, UNIVERSITY OF GLASGOW.

By JOHN H. TEACHER, M.A., M.B., C.M.

AMONG the graduates of the University of Glasgow who have become benefactors of their Alma Mater there is, perhaps, not one who more deserves the gratitude of his successors than the man who bequeathed to her the great museum and library which bears his name. William Hunter began his museum as a collection of anatomical and pathological preparations designed to supplement his dissecting room in the teaching of anatomy, which in those days included pathology. Years after, when he was the most successful practitioner of midwifery in London, he extended the plan of his museum, and collected anything and everything that might add to its usefulness in the diffusion of knowledge and culture.

Thus it came to embrace in addition objects of art, archæology, natural history, geology, mineralogy; it became, in fact, more than a museum for a medical school; he made it a university museum, and such a one as was unrivalled in its day by any other in Britain.

At first, doubtless, he collected for his own pleasure, but later it was certainly with a view to one day bequeathing it to trustees who might use it for the public benefit, and he chose the members of his old University as his trustees.

Of late years, however, the usefulness of the museum has been much impaired by the fact that the catalogues of the collections had become, through time and the number of additions

which had been made, both out of date and inadequate. Happily this fault has now for some years been in process of rectification, by the compilation and publication of new catalogues of various departments under the supervision of the keeper, Professor Young. Catalogues of part of the collection of coins¹ and of the anatomical and pathological preparations² have now been published; and catalogues of the library, both printed works and MSS., have been prepared, but cannot be printed in the meantime for want of funds.

The present article has been written at the request of the editors of the Glasgow Hospital Reports, who felt that an account of the overhauling and recataloguing of the Anatomical and Pathological Collection was a proper subject for their volume; that the more widely it was known that something new was being done for the museum in general the better it would be for that institution, and the more likely it was to be useful. Further, that this report would reach many graduates of the University and others whom the catalogues would not reach, and who, indeed, might not otherwise hear anything about this matter.

More particularly the present opportunity is taken to give what is not given in the catalogue of the anatomical and pathological preparations, viz., a general account of what that section of the museum contains, to indicate some of the points of special interest or value about it, and lastly a few things that might be done to increase its usefulness as a collection for teaching and for reference in scientific research. A full account of the work which, in the last five years, has been done in the way of overhauling and recataloguing the anatomical and pathological collection has been given in the preface to the recently issued catalogue of that section of the museum. A biography of William Hunter and a history of the origin and formation of his museum, and its relations to his written works, are also contained in the historical introduction.

¹ *The Stevenson Catalogue of Greek Coins in the Hunterian Collection*, by George MacDonald, M.A. Jas. MacLehose & Sons, Glasgow, 1899. A second volume is now in the press.

² *Catalogue of the Anatomical and Pathological Preparations of Dr. William Hunter in the Hunterian Museum, University of Glasgow*, by John H. Teacher, M.A., M.B., C.M. Jas. MacLehose & Sons, Glasgow, 1900.

The overhauling has meant remounting nearly all the preparations, and mounting many which had never been mounted before. It was no small labour; nor was the disturbing of the old preparations a matter to be undertaken lightly, but it has been managed without mishap, and it has greatly improved the appearance of the collection.

All the new catalogues are founded on those that were prepared during the lifetime of William Hunter, either by himself or by others working under his direction. The old MS. catalogue of the anatomical collection was published by the University in 1841; copies of the volume are not rare; it was sold at a shilling, and constituted a sort of perpetual admission ticket to the museum. In the new catalogue all that appeared to be valuable in the old has been embodied. At the same time great additions have been made in the way of altogether new or of more detailed descriptions, records of microscopic examinations, and references to and extracts from literature connected with the preparations—principally the writings of Hunter himself, but also those of John Hunter, Hewson, Cruikshank, and many others of less note. The collection has also been completely rearranged. The old order, where there was any attempt at order, was unsatisfactory; the rearrangement, which is naturally more or less provisional and temporary, brings the order of the collection into harmony with that in which the subjects illustrated are usually treated now; in this way, with the aid of the table of contents and index, it should be easy to find specimens of any kind that a person might wish to consult, or to say where a new specimen should be inserted. The museum copies are interleaved, so that descriptions of new preparations may be inserted as they are obtained. Thus, in these volumes there will always be descriptions of all the preparations in the collection, and the issue of a supplement when a sufficient number of these should have accumulated would be at any time a very simple matter.

The collection as now laid out contains about three thousand three hundred preparations—a collection of moderate size; but it is the quality of the preparations that is most remarkable. Nearly all of them have been beautifully finished, artistically mounted, and really illustrate what they were intended to show.

Further, although so many years have elapsed since they were made, the Hunterian preparations are as good as ever, and (this applies more particularly to the injected ones) as good as can be made now and a great deal better than we will give the time to make now. Such preparations do not bulk so largely in the teaching of anatomy and pathology as they did in Hunter's day, but they have still their sphere. The majority of them, used as he directed they should be used, are as valuable now as they ever were. The interpretation of the appearances which they show may change; they themselves are so "exactly Nature herself" that they can never get out of date. A better foundation for a great anatomical and pathological museum could not be desired.

As to the value of anatomical preparations there is considerable difference of opinion. William Hunter has left on record his ideas of their value, how they should be used, and the limitations of their usefulness.¹ The man who first made it possible for a British student to practise dissection of the human body without betaking himself to France or Germany, was not likely to imagine that preparations could take the place of the fresh subject; he taught his students that they must take the knife in their hands and learn to use it for themselves. "Preparations should not be used as substitutes for a body; but supplementally, to demonstrate such circumstances clearly as are intricate, confused, or invisible in the fresh subject. And a demonstrator who makes fine preparations should be very much upon his guard; otherwise he will be apt to make an abuse of preparations; he will insensibly contract a partiality to that in which he excels; the elegance of preparations is delusive with students; the more they are used, there will be less expense and trouble with fresh subjects." Properly used he had no doubt as to their usefulness, and "they serve two purposes, chiefly, to wit, the preservation of uncommon things, and the preservation of such things as required considerable labour to anatomize them so as to show their structure

¹"*Introductory Lectures*, delivered by Dr. William Hunter, to his last course of Anatomical Lectures, at his Theatre in Windmill Street: as they were left corrected for the press by himself." Published by his trustees. London. 4to. 1784.

distinctly. Of the first sort are the pregnant uterus, diseases, parts of singular conformation, etc. Of the second class are preparations of the ear, the eye, and, in general, such as show the very fine and delicate parts of the body, which we call the minutiae of anatomy."

By far the greatest part of the collection consists of wet preparations mounted in spirit. There are about three hundred mounted by a process which has almost disappeared nowadays, viz., dried and mounted in turpentine, which makes them transparent. This process was very useful for showing the vessels (blood or lymphatic) or ducts of injected organs, or such things as centres of ossification. There are nearly four hundred dry preparations, chiefly diseased bones, in jars, and a few large plaster of Paris casts, which are particularly interesting, as they were taken from the actual dissections which are figured in Hunter's "*Gravid Uterus*." There is also a fine set of embryological wax models, left to the museum by the late Dr. Allen Thomson. About sixteen hundred of the preparations may be classed as anatomical, and about the same number as pathological. Among the former division the series referred to in the passage quoted above may be mentioned as particularly good.

It is one of the special features of the collection that so many of the preparations are injected. The art of injecting—filling the vessels, whether blood-vessels, lymphatics, or ducts, with some fluid charged with fine pigment which would bring them clearly into view, either as an experiment in itself or as a preliminary to dissection—was one of the principal methods by which the sciences of anatomy and physiology were being advanced in Hunter's time. It can hardly be called a lost art, but it has been to a large extent laid aside in favour of other methods of research. For that purpose the methods by which William Hunter made his discoveries are worked out, or nearly so; but for teaching the elementary and at the same time fundamental part of anatomy—naked-eye anatomy—his preparations retain all their old value. To appreciate the advantages of injections so fine that they could reach the capillaries for preserving colour and fulness and the rounded outlines of soft and shrinking struc-

tures, one need only examine the beautiful specimens of the throat and nose or of the skin.

There is also a series which has quite a special value of its own. The lymphatic system can hardly be studied in the dissecting room on account of the impossibility, except in certain rare cases of disease, of making out anything beyond the glands, and one or two of the main vessels, since the demonstration of them involves injection by processes different from and more difficult than those used to exhibit the blood-vessels. We have in the Hunterian collection most of the original preparations by which the distribution of the lymphatic system was traced out by Hewson and Cruikshank—a most instructive series.

There is not much need for additions to the anatomical collection on the original lines, but what might with great advantage be added is a good set of the viscera prepared by the new formalin-glycerine process, which preserves the natural appearances so well.

The pathological division of the collection is not quite so satisfactory as the anatomical in one respect; many of the series are very weak in what may be termed the common objects of the *post-mortem* room, *e.g.*, there is only one preparation of secondary cancer of the liver. On the other hand, it contains a very considerable number of rarities. In particular there are many preparations which could not be obtained nowadays, and which we hope will never be obtainable again. For example, over a dozen preparations of septic osteomyelitis in amputation stumps, or in compound fractures, remind us of a time when sepsis was the rule after wounds, whether accidental or due to operation. There are also dozens of specimens of acute suppurative osteomyelitis which had not been operated upon. In those days it was the rule to leave them as far as possible to nature, and the preparations in the Hunterian Museum tell a tale of suppuration, the duration of which was limited only by the number of years the patient could stand it. It is a pity that we have the history of so few of these preparations.

The collection of calculi and concretions runs to about 330; for the most part they are of the common varieties,

but a number of them are exceedingly interesting. The series of monstrosities, though not very large, is very representative; it contains examples of nearly all the principal types of monstrosity. Comparatively few of them had been dissected, and as a rule no further dissection has been done except what was absolutely necessary for determining generally the nature of the malformation; but, in the future, specimens, after careful comparison with the undissected ones, might be dissected, so that the internal anatomy, as well as the external form, might be illustrated.

Lastly comes what is quite the finest part of the collection, viz., the obstetrical and gynaecological section. This is partly pathological, partly anatomical; it contains nearly six hundred preparations. The specimens illustrating the anatomy of normal utero-gestation are magnificent. A considerable number of them have been identified as the originals of the engravings in William Hunter's *Anatomy of the Human Gravid Uterus*, which is still the leading work of its kind in that department. There is hardly room for improvement in this particular series, and fresh examples of normal gestation or young abortions, instead of being turned into preparations, should be used for microscopical research after comparison with the preparations already here. In the two series, abnormalities and diseases connected with pregnancy and diseases of the female genital organs, there is ample room for additions. Through the kindness of various gentlemen a number have already been made, and more are particularly desired.

In this department of pathology the Hunterian Museum has no rival in Glasgow. In other departments it is out of the question for it to compete with the museums attached to the *post-mortem* rooms of the infirmaries; yet the value of a collection in the University beside the lecture rooms of surgery and medicine, from which preparations can be (and are) drawn to illustrate the lectures, is obvious. Further, it is no small advantage that the abnormal and the normal are here side by side for comparison.

The kind of preparations which it is most desirable to obtain are such as are related to some publication. They

form permanent tangible records of interesting cases, which it is most important to preserve; but only too frequently the difficulty of mounting them satisfactorily, and of keeping them and looking after them in the private house of a busy man, leads to their being spoilt or lost; whereas, were they placed in the museum of the University they would be well cared for, and would be always available for reference or inspection by workers on kindred subjects. A short *résumé* of the paper and the reference naturally would appear in the catalogue. Such preparations form a considerable, and perhaps the most valuable, part of all great pathological museums. It is an honour to a preparation to be admitted to the company of those made by the hands of Dr. William Hunter.

CAESAREAN SECTION AND ITS MODIFICATIONS, WITH AN ADDITIONAL LIST OF FIVE CASES.

By MURDOCH CAMERON, M.D., F.F.P.S.G.,
Regius Professor of Obstetrics and Gynaecology, Glasgow University ;
Physician for Diseases of Women, Western Infirmary ; Consulting
Obstetrician, Glasgow Maternity Hospital.

CAESAREAN section is an operation whereby an opening is made in the abdominal wall, and another in the uterus, through which the foetus is extracted.

According to Pliny, it is named Caesarean because the first of the Caesars was so extracted from his mother's womb as she was dying. According to another version it is named from the operation itself, *caeso matris utero*.

This operation was at first made upon dead women at a more or less advanced state of pregnancy. It is attributed to Numa Pompilius, one of the first kings of Rome, who enacted (*lex regia*) that a pregnant woman deceased could not be interred until the foetus was extracted. This law remained in operation throughout all countries under Roman rule, and was approved by the Church, as well as adopted as a civil law by Northern States, more especially Germany. For many years they dared not perform the operation upon a living woman, and in this way encouraged the performance of craniotomy, as the passage of the foetus through the pelvis in cases of deformity was impossible without mutilation.

Levret and Mauriceau deny that this operation was known to the ancients, but Dionis and Gardien refer to Pliny's *Natural History*. Dr. Mansfield published a work "On the Antiquity of Gastrotomy and Hysterotomy on the Living" (*Weber, das*

Alter des Bauch- und Gebärmutterschnitts am Lebenden zu Braunschweig, 1824).

He states that in an earlier work named *Mischnajoth*, written about 1400, there is this passage, "In a twin birth, neither the first child which by section of the belly is brought into the world, nor the one coming after, can attain the rights of heirship or priestly office."

Nicolai Falconiis recorded a case at Venice in 1491. The case of Jacob Niefer, the Swiss peasant who performed it upon his own wife, is frequently quoted, but most authorities are agreed that it was much later before it was generally attempted upon the living woman. In fact, we need only refer to the action of Mauriceau in the case treated by himself and Chamberlen, where the operation was delayed till after death, although Mauriceau was in actual attendance for several days. He wrote: "The child had been dead to all appearance about four days, and I told all the assistants that she could not be delivered. They asked me to perform Caesarean section, which I did not wish to do, knowing that it was always certain death to the mother." This poor woman died with her infant in utero twenty-four hours afterwards.

Rousset, physician to Catherine de Medicis, and contemporary of Paré, published a work upon the subject in 1581. This book was translated into Latin about ten years later. The author attempted to prove the possibility of saving the mother and child by means of this operation, but his views were opposed by Paré, Guillemeau, and others. In the middle of last century, the subject divided operators into two sections, the Symphyseans and Caesareans, or those who advocated division of the symphysis pubis and those who advocated Caesarean section.

It may be taken as a recognised rule in midwifery that no woman should be allowed to die undelivered without some attempt being made to save her and her offspring, or, at least to save her at the expense of her child.

Concerning the latter point, whether we are justified in destroying the infant when alive, there has been, and still exists, difference of opinion, due in some measure to religious belief and likewise to the personal feeling of the husband, who

felt that very little hope was held out to him that his wife could be saved by section. Amongst such men we had Napoleon, who, when appealed to by Dubois, said: "Treat the Empress as you would a shopkeeper's wife in the Rue St. Martin, but, if one life must be lost, by all means save the mother." In marked contrast to him we had Henry VIII., who, when thus questioned before the birth of his son Edward, exclaimed: "Save the child by all means, for other wives can be easily found." At the present time such men might be put down as either a good husband but a bad father, or a good father but a bad husband.

The doctrine of the Roman Catholic Church has been that, if you could not extract the child without killing it, you could not, without mortal sin, do so, and likewise until lately, it was held that the infant could not be baptised in the uterus, as it should be *natus* before it could be *renatus* by baptism.

Of late years the happy results following Caesarean section and Porro's operation have done much to efface the dreadful feeling, that we have got in such cases to decide whether the life of the mother or that of the child is to have our preference, seeing it is now quite possible to save both.

Barnes wrote: "Caesarean section is resorted to with a feeling akin to despair. Embryotomy stands first, and must be adopted in every case where it can be carried out without injuring the mother. Caesarean section comes last, and must be resorted to in those cases where embryotomy is either impracticable, or cannot be carried out without injuring the mother. There is, therefore, no election. The law is defined and clear. Caesarean section is the last refuge of stern necessity."

As against this statement, Dr. Barnes has recently said: "It is no longer permitted to us, without ample proof of clear necessity, to sacrifice the child in order to save the mother. The cases in which the two lives are supposed to stand in antagonism are vanishing before the light of modern science and skill."

If anything is needed to sicken one at the revolting practice of craniotomy, I might be allowed to relate the obstetric history

of a rachitic woman, who during her three last confinements was under my personal care :

1st—1862,	-	-	Embryotomy.
2nd—1863,	-	-	Embryotomy (labour induced).
3rd—1864,	-	-	Embryotomy.
4th—1865,	-	-	Induced labour at half term.
5th— — ,	-	-	Embryotomy (Birmingham, L.I. Hosp.).
6th—1868,	-	-	Induced labour at half term.
7th—1870,	-	-	Embryotomy.
8th—1871,	-	-	Embryotomy (eighth month).
9th—1873,	-	-	Embryotomy.
10th—1874,	-	-	Embryotomy.
11th—1875,	-	-	Induced labour at half term.

We must never forget that we have a sacred trust, and I hold we have no right to sacrifice a child, however unequal its life may be in some cases to that of the mother. In advocating the preference for section as against craniotomy in the living child, I do so only after very mature consideration, and with a feeling that to do otherwise would be to sacrifice a life which I was bound to preserve. I think the time has come when the lives of the mother and child may alike be saved, and prefer to think that an infant come to maturity is destined for something greater than to have its glimmering life extinguished by an accoucheur skilled in the use of a dreadful perforator. Let our motto be, "We live to save and not to destroy."

In another case where the obstetric history was like the preceding one, Caesarean section was performed, and the mother now attained her long wished-for desire, a living child.

Burns in twenty-four cases gave twenty-two deaths, whilst others gave the death-rate as from 50% to 100%.

With such results it is not to be wondered at that so many opposed the operation. In England, for example, accoucheurs condemned it absolutely. In Paris, during half a century, there was not a successful case, although it had been performed about sixty times. In the large Maternity Hospitals of Paris and Vienna with from four to eight thousand confinements in the year, not a single successful case of Caesarean section has been recorded. No doubt now exists but that the great fatality

was due to the fact that the operation was only resorted to after other measures had failed.

Indications for the operation.—As regards the general indications for the operation, of course they vary in the hands of different operators, as some, still looking upon Caesarean section as a last resource, divide the indications into absolute and relative. The absolute is where the deformity of the pelvis is so pronounced that the passage of even a mutilated foetus is impossible, whilst the relative is where they may remove a mutilated foetus by the natural passage with as good or a better result for the mother. It is here that difference of opinion exists. Baudelocque admitted Caesarean section in cases with a conjugate diameter under two and a half inches; Cazeaux two inches; Tarnier two inches, and Depaul from one and a half to two and a quarter inches where the child was alive, and under one and a half inch when the foetus was dead. Stolz advocated Caesarean section whenever the child was alive and could not be brought through the natural passage.

Scanzoni	-	-	-	-	under three inches.
Naegele	-	-	-	-	„ two „
Spiegelberg	-	-	-	-	„ „ „
Barnes	-	-	-	-	„ one and a half inch.
Playfair	-	-	-	-	„ „ „
Leishman	-	-	-	-	„ „ „

Of late years, the good results following Caesarean section in the hands of Cameron, Leopold, Sänger, and other operators has materially changed the views of many authors, who now favour Caesarean section more than they have done in the past.

Lusk, at the International Congress held at Washington in 1887, declared that Caesarean section was preferable to Embryotomy even with a conjugate diameter from two and a half to three inches when the child was alive.

It can well be urged that:

- (1) Embryotomy in a very contracted pelvis is as dangerous to the mother as Caesarean section.
- (2) Embryotomy always compromises the life of the child, whilst Caesarean section gives a living child.
- (3) No one has any right to sacrifice a child where he can save it, without exposing the mother to any additional risk.

For these reasons the operation should be one of election when the child is alive, and it should be performed before the patient is exhausted: in fact, early after labour has commenced, or even at full term before labour sets in, especially in multiparae. In all cases it should be done before rupture of the membranes, and if possible the patient should be placed under the care of an experienced operator.

Little difficulty is experienced in obtaining the consent of the patient and her friends, and it is better to have her under observation previous to the operation, so as to regulate her diet, and have her prepared for operation beforehand.

A very important point in favour of Caesarean section is that the Fallopian tubes can be tied and divided, so as to prevent subsequent conception, whereas Embryotomy may require to be performed ten or a dozen times.

Besides deformity of the pelvis, other conditions, such as tumours or cancer of the cervix uteri, may exist which would demand either Caesarean section or some modification of it.

If the child be dead and the conjugate diameter not over an inch and a half, Caesarean section should be done.

Rousset, the earliest writer upon this subject recognised the indications, the one furnished by the foetus, and the other by the mother. Under the first category he placed excessive size of the foetus, monstrosities, and faulty positions. Under the second he placed marked contractions from whatever cause. Some operators would include placenta-*prævia* and puerperal convulsions. Caesarean section might be advisable in some cases of eclampsia, but a skilful obstetrician would never think of such procedure in the case of placenta-*prævia*. In fact, the operators who advocate this step are surgeons who have little or no experience in obstetric practice.

Our decision for operation should be based upon the degree of contraction of the pelvis, the size of the child's head, and its reducibility, unless the obstruction is due to some other cause, such as cancer or the presence of a tumour in the pelvic cavity.

Every practitioner should be able to form a fair estimate of the amount of contraction, as it is easier to measure a contracted pelvis than a normal one, and it does not require

a highly skilled obstetrician to say before labour has commenced, or during the early stage of the process, that the diameter of the pelvis is, or is not less than three inches, and as a matter of fact, such a pronouncement should be within the skill of the ordinary practitioner, who should be more than a generally useful person, otherwise he will sink to the level of an ignorant midwife. Not only must he be able to form an estimate of the amount of contraction, but, by patient study of normal cases, qualify himself to form an opinion as to whether it will be impossible for a living child to pass, and also whether, under the difficult circumstances in which he may be placed, it would not be better to send the patient where Caesarean section could be safely performed than to extract a mutilated foetus through a minimum diameter.

With a diameter under two and a half inches, where engagement of the head is impossible, no one should hesitate to advise Caesarean section, although there will always remain cases such as where the child is dead or a subject of hydrocephalus, in which craniotomy may be resorted to.

Experience alone will enable one to avoid extreme measures in cases with a conjugate diameter measuring more than three inches, and where the skilled practitioner will weigh the chances between premature induction of labour and symphysiotomy.

There can be no questioning that Caesarean section is a highly dangerous operation, but the danger, it should be remembered, depends for the most part on delay, and death most frequently results not so much from the operation, as from previous operative abuse, which is the just term for all injudicious attempts to extract the foetus through a deformed natural passage.

Success depends upon prompt interference before the patient is exhausted, as then there is less danger from haemorrhage, delayed shock, or peritonitis.

When Caesarean section has been resolved upon, another question presents itself, namely, whether Caesarean section or Porro's operation is preferable. If the former, there still remains to be decided whether the operation will be

accompanied or followed by a removal of the ovaries, or the patient be sterilised by the simple expedient of tying and dividing the Fallopian tubes. This we have done in about fifty cases and no harm has resulted, although theorists would have us believe that such a procedure would be surely followed by hæmatocele. Where there is a choice of operation, Caesarean section is to be preferred, as it can be completed much sooner, and is free from the danger of shock and peritonitis which might complicate Porro's operation.

The preparation of the patient will depend upon the urgency of the case. When she is under observation, it is better to confine her to bed for a couple of days beforehand, and the bowels should be moved by an enema and a slight laxative. The abdomen is washed and gently scrubbed, and the parts shaved, whilst the vagina is cleaned and rendered aseptic. The preparation in fact is the same as in any other abdominal section. The operator and his assistants who have to do with the case must be exceptionally careful in cleansing and disinfecting their hands, whilst the chief nurse should see that the instruments and sponges are sterilised and counted.

Very few instruments are necessary, and should comprise two straight scalpels and one blunt-pointed bistoury, pressure forceps, dissecting forceps, scissors, director, twenty straight two and a half inch Hagedorn needles, compression pessary, aseptic silk, silk-worm gut, adhesive plaster, and dressings.

The catheter should always be passed into the bladder shortly before operation. The needles should be threaded in pairs beforehand, with No. 3 Chinese twist silk ligatures, about twenty inches long, and placed in a towel wrung out of 1-30 carbolic solution, ready for use.

Palpation will reveal the position of the foetus, and this is all the more important, as from this you will know the attachment or site of the placenta.

Briefly, my experience in Caesarean section has shown me that in dorso-posterior positions the placenta is attached upon the anterior wall, whilst in dorso-anterior positions the placenta is upon the posterior wall.

(a) Thus, in the first cranial position, or O. L. A., the placenta will be found upon the posterior wall, and somewhat to the right side.

(b) In the second cranial position, or O. D. A., the placenta will be upon the posterior wall, and somewhat to the left side.

(c) In the third cranial position, or O. D. P., the placenta will be upon the anterior wall, and somewhat to the left side.

(d) In the fourth cranial position, or O. L. P., the placenta will be upon the anterior wall, and somewhat to the right side.

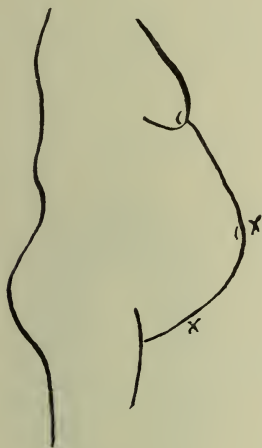


FIG. 1.



FIG. 2.

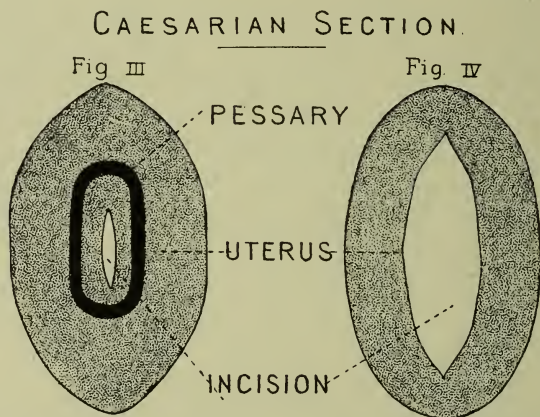
The foetus and placenta will be found in the same relation in the various pelvic positions.

From this knowledge you know when your uterine incision is likely to cut down upon the placenta, and you also form an idea as to how you will extract the foetus. The abdominal incision should be made in the median line, as in ovariectomy, and it will vary in situation according to the distension of the abdominal wall.

Thus, if the abdomen takes the form as seen in Fig. 1, an incision of from five to six inches may be got without extending beyond the umbilicus, but when it is pendulous as in Fig. 2, the incision must of necessity extend more or less above the umbilicus.

Before opening the uterus, the operator should satisfy himself that the uterus is not only in the median line, but that it is not twisted upon its axis. This is found by locating the position of the Fallopian tubes by means of the fingers. He will frequently find the left tube more or less in front, as the uterus is usually rotated to the right. This displacement must be corrected, and if necessary an assistant can easily keep the uterus in position by pressing with his hand on the right side.

When the placenta has its attachment upon the anterior wall, the site is seen to bulge, and upon palpation has a fluctuant feeling akin to a large pointing abscess.



The next point is to open the uterus with as little loss of blood as possible, and this can be done by placing a flat vulcanite pessary upon the uterine wall around the point to be incised, as in Fig. 3.

The operator with the fingers of his left hand applies pressure upon the pessary, whilst his assistant does the same on the opposite side. The incision is then made with two or three strokes of the scalpel, and the blood sponged away by the assistant with his right hand. After this has been done, no more bleeding takes place unless the placenta is attached in front, as the pressure with the pessary thoroughly prevents

even oozing. Care should be taken not to puncture the membranes, which will soon be observed and recognised by their pearly colour. If the placenta intervenes, this method of pressure is beneficial not only in preventing bleeding, but also in permitting us to observe its tissue, which is recognised by its darker colour.

Whenever the membranes are reached, a director is placed within the opening, which is then enlarged with a blunt pointed bistoury, upwards and downwards as far as the pessary will admit. At this stage the compression pessary is removed, and the incision extended upwards and downwards sufficiently to permit the passage of the foetus. The extension of the incision downwards should be limited, as it is likely to interfere with proper contraction of the uterus. Should the placenta intervene, it must be dealt with as a placenta praevia after completing the incision, that is, either separated upon one side or, if central, pierced by the hand. There must be no hesitation in extending the incision, which is made upwards and downwards from within outwards in each direction with a blunt pointed bistoury, so as to make an incision of about five or six inches, as in Fig. 4. The left hand is inserted without rupturing the membranes till the head is being turned out, or the feet grasped, and then the child should be extracted without delay. On no account should the hand be withdrawn after its insertion, unless during extraction of the foetus, as the uterus speedily contracts. If the shoulder presents, a hand should be placed upon it to prevent its expulsion, as it adds very much to the difficulty when any portion of the child's body is allowed to protrude.

The child having been extracted, the assistant places a large flat sponge over the upper angle of the abdominal incision, to prevent the bowels from escaping, and then with both hands grasps the uterus, so as to prevent bleeding.

The cord having been tied and divided, the placenta is immediately removed with the left hand, great care being taken to secure the removal of all membranes and to prevent the entrance of blood into the peritoneal cavity. The assistant now everts the uterus from the cavity, and pushes a flat sponge behind it. The lips of the uterine wound are next

everted, the assistant grasping the upper angle and wall with his right hand, and the lower angle and wall with the left, as in Fig. 5.



FIG. 5.

The operator immediately inserts the silk ligatures, beginning at the middle, each suture grasping the entire wall, with the exception of the mucosa. From seven to ten sutures should suffice, as with the contraction of the uterus the incision is greatly diminished.

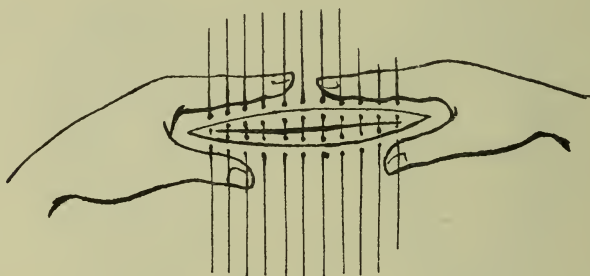


FIG. 6.

This accomplished, the sutures are gathered up and a large flat sponge laid over the anterior wall, and another behind. Firm compression or kneading is then made through the sponges, with the result that the uterus contracts firmly. The assistant should again seize the uterus as before, whilst the operator ties the sutures. When this has been accomplished, the whole organ is enveloped in a large, warm, flat sponge, and firm compression again made so as to ensure thorough contraction. Should any oozing appear at the needle punctures, a second warm sponge should be applied, and very slight compression will suffice to overcome any tendency

to relaxation. Should the peritoneal edges gape at any points, a few superficial fine sutures should be inserted to bring the margins together.

The performance of hysterectomy for bleeding is bad treatment, and indicates that the operator has lost his nerve, as pressure with a warm sponge with both hands never fails to secure thorough contraction.

Several operators advise the introduction of a drainage tube through the cervix and vagina, and the leaving it there to act as a drain. Nothing could be worse. Of course, it is the procedure of a surgeon, but everyone who has practised midwifery knows that the presence even of a clot in the uterus may lead to serious haemorrhage. Such a body as a tube, if not expelled, would induce haemorrhage, distension of the uterus, and bursting of the incision, with speedy death of the patient. This is no mere theory, but is what has actually taken place where drainage has been resorted to. On no condition should the uterine cavity be washed out or medicated in any way. The less the parts are interfered with the better.

After the ligatures have been cut short, the next step is to ligature and divide the Fallopian tubes with aseptic silk, in order to prevent future conception. Of course, the consent of the patient for this procedure should have been obtained beforehand. Two ligatures are tied upon each tube, which is then divided between those points. This method is effective, and leads to no complications nor bad results, nor is menstruation interfered with. Next the cavity is cleaned by the removal of all clot, etc., and the uterus replaced. The external wound in the parietes is closed in the usual way with silk-worm sutures. The vagina should now be cleansed of all clot and sponged out, after which an antiseptic pad should be applied to the vulva.

The wound should be dusted with iodoform, and a few layers of gauze placed over the wound. This should be secured with plaster, both to prevent slipping of the dressing and strain on the sutures, in case of sickness or cough. A sheet of Gamgee is next applied, and then the bandage.

The after treatment consists of sips of warm water, say a tea-spoonful every fifteen minutes for twelve or twenty-four hours, after which milk and soda may be given in increasing quantities. For a few nights, a half-grain morphine suppository is given. The urine should be drawn off every six hours for two or three days, care being taken to thoroughly cleanse the parts before doing so.

On the fourth day, two tea-spoonfuls of glycerine in a couple of ounces of soapy water is administered as an enema, and if necessary some slight aperient by the mouth. The bowels having been moved, the patient is allowed chicken soup, fish, eggs, beef-tea, etc. If the child is to be nursed, it may be put to the breast on the second or third day.

The abdominal sutures may be removed in from ten to fourteen days, and the patient allowed to rise at the end of four weeks. She should always wear an abdominal belt, and should be warned against kneeling when scrubbing floors, as this is apt to induce hernia from pressure and stretching of the cicatrix.

In review, it may be explained that rupture of the membranes, either intentionally or by labour, means a contraction of the uterine wall, and as a consequence a greater wounding of the uterine tissue, in order to secure a sufficient opening to extract the child. Some operators, instead of using manual or pessary compression to prevent bleeding when opening the uterus, make use of an elastic ligature. The uterus is first everted, and the elastic ligature then passed round the cervix. This not only necessitates a much larger abdominal incision, but also induces asphyxia of the foetus, and causes inertia of the uterus, as the organ does not so readily respond to kneading. Its use is therefore conducive to haemorrhage. Veit, Doleris, and Pajot have blamed it for causing death from haemorrhage, and Zweifel, Sanger, and Lusk have also noticed this complication.

Caruso advised the early removal of the ligature.

Another way of dealing with the uterine incision is by Sanger's method. In this procedure, the muscular wall of the uterus is closed with from ten to fifteen sutures which approximate to, but do not include the mucosa, and between each

suture two superficial sutures are inserted to unite peritoneum to peritoneum. Previously, the peritoneum was separated from the muscularis, and a wedge-shaped piece of muscularis removed from each side, the base of the wedge being outermost. This done, the peritoneal flaps were folded into the wound and secured by the superficial stitches. Such a detailed process is quite unnecessary, as the sutures, as recommended by Cameron, secure perfect apposition, not only of the muscular tissue, but also of the peritoneum. In fact, most operators now make use only of eight or ten deep sutures, and reserve superficial sutures to secure contact where there is any gaping between the stitches. Such unevenness can be readily avoided by beginning in the middle and working towards each end, and by taking care to keep the sutures at regular intervals.

Porro's modification. The fatal results following the early Caesarean section led to a modification of the operation. It had been found by experiment that the uterus in pregnant rabbits could be removed with better results than by simple section, and therefore it was concluded that similar results would follow in the case of women.

Blundell, in writing upon this subject, said such a method might prove an eminent and valuable improvement, but he also wrote, in speaking of deaths from peritonitis after Caesarean section, that experience sometimes contradicts our most favourite opinions, and that something of the kind would be found to occur in the cases under consideration, as he had no doubt the risk of diffused peritonitis had been greatly exaggerated. How his surmise has proved true, is seen in the present-day position of abdominal surgery.

Acting on the lines suggested, Storer of Boston in 1868 first practised amputation of the uterus after section. The case was one of pregnancy complicated with a fibroid of the uterus. He was interrupted by such an alarming haemorrhage that he had to remove the body and fundus with the ovaries, but his patient died three days afterwards. This was an operation of necessity.

Porro first performed the operation as a matter of choice, as he considered it impossible to secure the uterine incision in Caesarean section, so as to prevent the flow of blood and septic

fluid into the peritoneal cavity. The results got under anti-septics in other abdominal operations encouraged him to make the attempt, and in 1876 he did so with happy results. Others took up the operation, and very quickly the old Caesarean section was superseded by it, but only for a few years, as now Caesarean section can be performed without the slightest danger from bleeding, peritonitis, septicaemia, or other dangers that Porro's operation sought to avert.

At the present day, Porro's operation is an operation of exception, that is only necessary in some conditions, such as serious rupture of the uterus, or where labour is obstructed by a large fibroid. As regards the steps of the operation, it is to begin with similar to Caesarean section. It is only after the uterus has been emptied that it varies, as at this point the uterus is everted and an elastic ligature applied round it, just above the os internum.

The uterine tissues are then compressed until the bleeding has ceased. The uterus is then removed, and the stump secured outside the abdominal wound, and maintained in position by needles and a serrenoeud.

Porro, upon emptying the uterus, transfixes it with a trocar and canula at the union of the body and cervix. He then withdraws the trocar, and passes two silver wires through the canula, which was also withdrawn and the wires tied, one upon the right and the other upon the left side, including in their grasp the ovaries and tubes. This done, the uterus and appendages above the wires were cut away, whilst the stump was secured outside. The method has been improved by transfixing with needles and ligating with a serrenoeud instead of with separate wires.

The stump is dusted with iodoform, and dressed with gauze all round. The needles should be raised to allow of proper packing. A layer of sublimated Gamgee should be placed over all. It may require to be dressed daily, and the ligated portion usually separates about the tenth day, but the raw cavity requires regular dressing till perfectly healed.

It was urged as an important factor that Porro's operation prevented future conceptions, but this end is gained in Caesarean section by the more simple method of tying and dividing the tubes.

Some operators now prefer to remove the entire uterus, where a fibroma or a myoma complicates the condition, and such is my practice. The following case illustrates the method adopted in such a complication :

Mrs. C., aged 36, was admitted to the Western Infirmary, under my care, on July 16th, 1895, complaining of great distension of the abdomen. The patient considered herself about five months pregnant, but as the abdomen was so much distended she consulted me regarding her condition. She stated that she had always enjoyed the best of health until the spring of the present year, that menstruation commenced at the age of 15 and continued perfectly normal and regular until February, 1895, that she had been married for about two years, and had had no children and no miscarriages.

About eight years ago the patient noticed for the first time, and quite by accident, a swelling in the left iliac region. It was about the size of a hen's egg when she first felt it. For a month or two it increased in size slightly, then ceased growing, and remained stationary until she became pregnant at the end of February. On February 19th she menstruated for the last time. Three or four weeks afterwards she began to be troubled with morning sickness, and about the middle of June she felt foetal movements for the first time. She was quite satisfied that she was pregnant: but what troubled her was the fact that her abdomen became so rapidly distended. It was, however, during the six or seven weeks previous to her admission into hospital that the distension seemed to increase most rapidly.

Her general condition on admission was not very satisfactory. There was great oedema of the legs and thighs, but apparently no ascites. There was some dyspnoea. The mucous membranes were of a good colour. She had no difficulty in passing urine, but her bowels had been very constipated. There was nothing abnormal in the lungs and heart: milk could be expressed from both breasts. The abdomen was much distended, presenting the appearance of a pregnancy at full time. It measured 37 inches at the level of the umbilicus. On palpating the abdominal tumours, a large smooth, round, hard swelling was found to occupy the left side. It crossed the middle line, and was continuous with a swelling on the right

ADDITIONAL LIST OF DR. MURDOCH CAMERON'S CASES OF CAESAREAN SECTION.

No.	Date.	Age.	Number of Pregnancy.	Diameter of Conjugate.	Duration of Labour.	Results.		Remarks.
						Mother.	Child.	
39	Dec. 9th, 1897,	34	9th	2 Inches.	2 Hours.	Alive	Alive	Fallopian Tubes Tied.
40	June 6th, 1898,	27	3rd	2½	2	Alive	Alive	Do. do. do.
41	Jan. 4th, 1899,	28	2nd	2½	3	Alive	Alive	Do. do. do.
42	Apr. 13th, 1899,	34	5th	2¾	6	Alive	Alive	Do. do. do.
43	Octr. 12th, 1899,	32	5th	2	3	Alive	Alive	Do. do. do.

side, from which it was separated, however, by a sulcus. This swelling on the right side was elastic in consistence, but two or three hard rounded nodules could be distinctly made out upon its anterior surface. The uterine *souffle* could be heard over it, but no foetal heart sounds could be recognised.

On vaginal examination, the whole cavity of the pelvis was filled up with a hard mass firmly impacted into it, and continuous with the tumour above. So completely did this tumour block up the pelvic cavity that the forefinger could with difficulty be passed up the vagina. The uterus appeared to be very much drawn up, as the examining finger could not reach the os externum.

On July 19th the patient was examined under chloroform, but nothing further was made out.

On July 22nd abdominal section was performed. On opening into the abdomen a large irregular tumour was met with, the left portion consisting of an interstitial myoma, in the right of the distended uterus. In addition, implanted on the upper and posterior uterine walls, were several subserous myomata, varying in size from a walnut to an orange. Having made an incision through the anterior wall, a foetus of 5 months was removed. It was perfectly formed, and had evidently only quite recently died. The placenta was well developed, and was easily detached. An elastic ligature was passed round the uterus and tumours as close as possible to the cervix and secured, after which the uterus with the tumours were cut away. The uterine and ovarian arteries could now be more easily reached, and were ligatured. The ovaries and tubes were also removed. The tumour blocking up the pelvis was with some difficulty drawn up on account of impaction and adhesions. Having enucleated this mass of tumour nothing remained but the cervix, which was also separated from its connections. The peritoneum was stitched round and round with catgut sutures to the mucous membrane of the vagina. These sutures were then drawn down into the vaginal canal by means of a pair of long forceps passed up into the abdomen through the vagina. The abdominal wound was then closed with silk-worm sutures; no drainage tube was used. The parts removed weighed 27 lbs.

The patient made a good recovery. She had a little sickness on the third and fourth days, when the temperature thrice registered 100.2° F. After that it never rose above 100° , and fell to normal after the ligatures were discharged from the vagina on the tenth to the twelfth days. She left the hospital in the seventh week after the operation perfectly well.

The description given beforehand may be applied to all these cases so far as regards the operation, but it may be mentioned that the infant in the fourth case developed icterus of a severe type. Purpura spots appeared on the face, and the child rapidly got weaker and died the following day.

One point of note happened in the fifth case, namely, a slight difficulty was experienced in removing the after-coming head of the child from the uterus owing to its being grasped, not by the uterine wound, but by the retraction ring. This complication was easily overcome, but as I have never heard or read of such a fact, I here state it, recognising that such a contraction might readily form a delay during the second stage in a normal labour.

STATISTICS OF THIRTY YEARS' WORK (1869 TO 1898 INCLUSIVE) IN THE OUTDOOR AND INDOOR DEPARTMENTS OF THE GLASGOW MATERNITY HOSPITAL.

By ROBERT JARDINE, M.D., ETC., ETC.,

Physician to the Glasgow Maternity Hospital, Examiner in Midwifery to the University of Glasgow, President of the Glasgow Obstetrical and Gynæcological Society.

THE following statistics have been compiled from the Journals of the Glasgow Maternity Hospital. They represent the full-time and premature labours attended in the outdoor and indoor departments of the hospital for 30 years, from 1869 to 1898 inclusive. The abortions and miscarriages have not been included, only labours with viable children. Three periods of 10 years are dealt with in each department. The 30 years' work of each department is combined in two tables, and finally the whole work is summed up in one table. The labours are classified according to their numbers from 1 to 20, and the ages of the patients are divided into 5-year periods beginning at 15 and ending at 55. The married and single have been kept separate. The fatal cases are tabulated in the small columns to the left of the others. I have had to omit perhaps 500 or 600 cases where the age or number of the confinement has not been noted, but none of these were fatal ones. The causes of death are recorded under two headings—(1) Causes primarily due to parturition, and (2) Causes not primarily due to parturition. Under the first heading I have included the few cases in which no cause of death was given. Some of these probably belong to Class 2, but their number is so small that they do not much influence the final result.

The operative cases and the serious complications have also been tabulated. The fever cases and some of the septicaemia ones were transferred to the Fever Hospital. Through the kindness of Dr. Dick, a former Maternity resident, I have been able to trace nearly all the septic cases, especially those of the last 10 years. The fatal ones have been included in the lists. The typhus and scarlet fever cases I have not traced, but as they are diseases unconnected with parturition their omission is of small consequence.

The records of the cases have on the whole been faithfully kept, but of course there are bound to be some errors. I have been most particular about the fatal ones, and they are the ones which would be sure to be recorded. As the entire work has been done by myself I can speak with some confidence as to the tables being as nearly accurate as one can get them.

To discuss the cases properly an entire volume would be necessary, but as space is limited I shall merely point out a few of the outstanding features and leave the tables to speak for themselves.

The outdoor cases are the more valuable of the two, as the conditions under which the work is done more nearly approach those of ordinary private midwifery work than those of the indoor. The patients are attended at their own houses by nurses, students, and young graduates, who do their work under the supervision of the outdoor house surgeons. The abnormal cases are dealt with by the house surgeons and district accoucheurs, and exceptionally difficult cases are transferred to the hospital or are dealt with at home by one of the chief physicians.

A study of the tables brings out some very interesting facts. We shall take the outdoor ones first.

	No. of Cases.	Total Fatal.	Approximate Proportion.
1st decade,	8,553	60	1 in 142
2nd ,,	12,402	72	1 in 172
3rd ,,	20,156	56	1 in 360
	<hr/> 41,111	<hr/> 188	1 in 218

This shows a remarkable fall in the death-rate.

If we take the deaths due primarily to parturition, we see a much more striking improvement.

	No. of Cases.	Fatal.	Approximate Proportion.
1st decade,	8,553	46	1 in 186
2nd ,,	12,402	52	1 in 238
3rd ,,	20,156	39	1 in 517
	<hr/> 41,111	<hr/> 137	1 in 300

Of these 137 deaths, 46 were due to septicaemia, viz. :—

	No. of Cases.	Fatal.	Approximate Proportion.
1st decade,	8,553	18	1 in 475
2nd ,,	12,402	14	1 in 886
3rd ,,	20,156	14	1 in 1440
	<hr/> 41,111	<hr/> 46	1 in 894

This is a most gratifying result, because it has been frequently pointed out that puerperal septicaemia throughout the country generally is almost if not quite as fatal as it was before the introduction of antiseptics. In our next ten years I am convinced that there will be a much greater improvement.

If we consider the total cases (Table IV.) we find there were 41,111 with 188 deaths (1 in 218): of these 137 or 73 per cent. were primarily due to parturition (1 in 300) and of these latter 46 or 25 per cent. were due to septicaemia (1 in 894). Taking the married and single separately we find 37,338 married with 164 deaths (1 in 227), and of those primarily due to parturition 118 or 72 per cent. (1 in 316). Of the 3773 single ones 24 were fatal (1 in 157) and 19 or 79 per cent. primarily due to parturition (1 in 200). As one would expect, the death-rate among the single is considerably higher than among the married.

It would be interesting to study the death-rate according to the age and number of confinement, but we cannot go into that at present. I merely wish to point out that the rate is very high in primiparae (1 in 145 among the married and 1 in 137 among the single), and it then falls among the married until we reach the 11th labour, when there is a very marked rise; the 13th comes next, and then the 12th. Among the single, 19 out of the 24 deaths occurred among the primiparae.

TABLE I.—SINGLE (OUTDOOR), 1869-78.

	15-19	20-24		25-29	30-34		35-39	40-44	45-49	D.	A.	Total.	GRAND TOTAL.—M. & S.	
													D.	A.
1	1	258	3	210	1	52	1	1		6	546	552	18	1844
2		8	1	105		43				1	175	176	7	1374
3				23		26		1			61	61	3	1111
4				13		14					34	34	4	964
5				4		6		1			19	19	7	779
6						6			1		15	15	4	711
7						4					4	4	3	562
8											4	4	2	388
9													3	334
10														199
11													6	144
12													1	82
13													1	39
14													1	12
15														7
16														2
17														1
	1	266	4	355	1	145	1	59	3	7	854	861	60	8553

Operative Cases, Outdoor, 1869-1878:—

	COMPLICATIONS.		FATAL.	
Forceps, -	-	-	-	-
Version, -	-	-	-	-
Craniotomy, -	-	-	-	-
Primary Postpartum Haemorrhage,	34	3	3	3
Secondary	7	5	5	1
Placenta Praevia, -	-	-	-	-
Accidental Haemorrhage, -	-	-	-	-
Eclampsia, -	-	-	-	-
Rupture of the Uterus, -	-	-	-	-

(a) The result to the 2nd case of smallpox is not stated. The child died shortly after birth.

(b) The two cases of typhus were removed to the fever hospital. Result not stated.

There were three cases of triplets: two in 1869 and one in 1871.

TABLE II.—MARRIED (OUTDOOR), 1879-88.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	D.	A.	Total.
1	3	9	2	1	1	6	2		16	1733	1749
2		2	4	1	21	8			7	1665	1672
3		2	2	1	49	8			5	1630	1635
4		2	2		117	11	1		4	1369	1373
5		2	1		127	24	1		2	1114	1116
6			4		190	35	2			936	941
7					238	1		1	3	768	771
8					241	32	7	1	8	618	626
9				5	222	61	5		3	475	478
10					172	2	5		4	303	307
11					86	60	8		2	180	182
12					47	53	5		1	112	113
13					27	19	2		1	54	55
14					6	11	3	1		21	21
15					3	3	2			8	8
16					2	1	1			4	4
17						3				3	3
18										1	1
19					1		1			2	2
20					1					1	1
	3	15	20	10	12	6	46	3	61	10,997	11,058

Causes of death among the Outdoor Cases from 1879 to 1888 :—

I. Causes primarily due to parturition—
 (a) One of these occurred after placenta prævia, and one after accidental hæmorrhage.

(b) Three others died: one of septicaemia, one of bronchitis, one of pneumonia.

(c) One other died of septicaemia.

II. Causes not primarily due to parturition—

1. Pneumonia, - - - - -
2. Phthisis, - - - - -
3. Bronchitis, - - - - -
4. Cardiac Disease, - - - - -
5. Lingering Disease, - - - - -

	Single.	Married	Total.
1.	1	3	4
2.	1	6	7
3.	0	3	3
4.	0	3	3
5.	0	3	3
	-2	-18	-20

	Married.	Single.	Total.
1. Septicaemia, - - - - -	11 (a)	3	14
2. Primary Postpartum Hæmorrhage, - - - - -	0	0	0
3. Secondary " - - - - -	0	0	0
4. Placenta Prævia, - - - - -	8 (b)	0	8
5. Accidental Hæmorrhage, - - - - -	1 (c)	0	1
6. Eclampsia, - - - - -	4	5	9
7. Rupture of Uterus, - - - - -	0	0	0
8. Shock, - - - - -	6	0	6
9. Embolism, - - - - -	2	1	3
10. Cause not stated, - - - - -	2	0	2
	-43	-9	-52

TABLE II.—SINGLE (OUTDOOR), 1879-88.

15-19		20-24		25-29		30-34		35-39		40-44		D.	A.	Total.	GRAND TOTAL.			
															D.	D. & A.		
5	417 27 2	4 1	441 154 20 3	1	62 55 36 18 7 1		11 15 14 8 5 4 3 1 1 1		8 2 3 3 1 3 2 2 1		1	9 2	940 253 75 33 13 8 3 3 3 1 1	949 255 75 33 13 8 3 3 3 1 1	25 9 5 4 2 2 5 3 3 3 4 2 1 1 1	2698 1927 1710 1406 1129 949 774 629 481 308 183 113 55 21 8 4 3 1 2 1	72	12,402

Operative Cases, Outdoor, 1879-88—

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	
Forceps, -	518	1 in	24		
Version, -	116	1 in	107		
Craniotomy, -	20	1 in	620		
			FATAL.		
Primary Postpartum Haemorrhage,	96	9			
Secondary "	4	0			
Placenta Praevia, -	42	11			
Accidental Haemorrhage, -	47	2			
Eclampsia, -	24	9			
Rupture of Uterus, -	0	0			
Inversion of Uterus, -	0	0			

THE CASES, ACCORDING TO THE SITE OF THE LESION.	No. of Cases.	COMPLICATIONS.			FATAL.
		1 in	24	1 in	

TABLE III.—MARRIED (OUTDOOR), 1889-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	D.	A.	Total.
5	692	2	2								
1	179	2	1					1	9	2311	2320
2	20	1	3	1					3	2710	2713
3	2		1003	279	23	5			5	2457	2462
4			1218	1	89	15		1	3	2328	2329
5			1	1	131	18			1	2010	2017
6			1	1	206	26		1	7	1755	1760
7			1	1	302	48			5	1452	1459
8			1	3	384	67			2	1201	1203
9			1	3	480	91		1	3	860	863
10			1	1	433	109			1	619	620
11			1	1	309	142			6	388	394
12			1	1	196	138	1		1	221	222
13			3	12	98	98	10			118	118
14			3	8	56	53	6			63	63
15				4	22	31	1			25	25
16				2	6	16	2			10	10
17					3	5				3	3
18						3				6	6
19					1	5	1			1	1
+5	893	6	4510	11	2792	4	1	3	50	18,538	18,588

Causes of death among the Outdoor Cases from 1889 to 1898 :—

I. Causes primarily due to parturition—

1. Septicaemia, - - - - -
2. Primary Postpartum Haemorrhage, - - - - -
3. Secondary " - - - - -
4. Placenta Praevia, - - - - -
5. Accidental Haemorrhage, - - - - -
6. Eclampsia, - - - - -
7. Rupture of the Uterus, - - - - -
8. Shock, - - - - -
9. Embolism, - - - - -
10. Persistent Vomiting, - - - - -

(a) One of these was after postpartum haemorrhage, and one after placenta praevia.
 (b) After craniotomy.

II. Causes not primarily due to parturition—

1. Pneumonia (a), - - - - -
2. Phthisis, - - - - -
3. Bronchitis, - - - - -
4. Cardiac Disease, - - - - -
5. Acute Gastritis, - - - - -

(a) One after Eclampsia.

TABLE III.—SINGLE (OUTDOOR), 1889-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	D.	A.	Total.	GRAND TOTAL.	
											D.	D. and A.
1	2	470	117	20	4	1	1	4	1099	1103	13	3423
2	26	457	73	28	3	1		1	305	306	4	3019
3		174	28	11	2				89	89	5	2551
4		48	14	9	2	1			38	38	1	2367
5		12	7	5	2				14	14	7	2031
6			2	2	2				7	7	5	1767
7		1	2	3	3	1			7	7	7	1466
8				1	2			1	2	3	3	1206
9									1	1	3	864
10											1	620
11											6	394
12											1	222
13												118
14												63
15												25
16												10
17												3
18												6
19												1
	2	496	3	722	241	79	20	3	1	1	6	1562
												20,156

Operative Cases, Outdoor, 1889-98—

Forceps, -	759	1 in	26	FATAL.
Version, -	129	1 in	156	6
Craniotomy, -	16	1 in	1260	0
Induction of Labour, -	3	1 in	6718	0
COMPLICATIONS.				FATAL.
Primary Postpartum Haemorrhage,	156		6	0
Secondary " "	3		0	0
Placenta Praevia, -	33		7	0
Accidental Haemorrhage, -	72		6	0

COMPLICATIONS.

Eclampsia, -	25	FATAL.
Rupture of the Uterus, -	0	6
Inversion of the Uterus, -	0	0
Insanity, -	1	0
Chorea, -	0	0

In one case, act. 28, iv.-para, the woman had never menstruated.
 In one case the hymen persisted and had to be incised.
 One of the cases of placenta praevia had twins.
 Three patients were sent to Belvidere suffering from septicaemia.
 They all died, and are included among the deaths from that cause.

TABLE IV.—MARRIED (OUTDOOR), 1869-98.

15-19		20-24		25-29		30-34		35-39		40-44		45-49		50-54		55-59		D.	A.	Total.
1	10	1320	17	2902	5	661	4	161	1	60	16	4			1			37	5324	5361
2	1	334	5	3363	6	1386	3	356	1	103	23	1						16	5567	5583
3		39	3	2025	7	2218	3	643		178	30	1						13	5134	5147
4		5	2	859	3	2367	4	1000		343	48	1						9	4623	4632
5			1	235	7	1799	3	1332	3	434	71	6						16	3877	3893
6				50	5	1050	5	1509	4	645	120	8			1			14	3383	3397
7				16		451	6	1351	4	804	144	8			1			13	2775	2788
8				5	1	180	6	923	3	863	216	16			2			12	2205	2217
9				1		65	2	528	7	814	241	15			2			9	1666	1675
10						15		246	3	574	264	21			1			5	1121	1126
11					1	8	1	83	6	330	263	20	2		2			14	706	720
12						6		26	1	169	187	26						3	414	417
13						1		16	2	95	1	4			2			3	210	213
14						1		4		28	49	10				1			95	95
15								2		10	24	4							40	40
16									5		7	4							16	16
17											6	1							7	7
18									1		5	1							7	7
19									2			1							3	3
20												1							1	1
	11	1898	28	9456	35	10,208	37	8180	35	5458	16	1808	2	153	12	1	164	37,174	37,338	

Causes of death among the Outdoor Cases from 1869 to 1898 :—

II. Causes not primarily due to parturition—

	Total.	Married.	Single.
I. Causes primarily due to parturition—			
1. Septicaemia,	46	38	8
2. Primary Postpartum Haemorrhage,	17	16	1
3. Secondary "	5	5	0
4. Placenta Praevia,	17	17	0
5. Accidental Haemorrhage,	10	9	1
6. Eclampsia,	20	14	6
7. Rupture of the Uterus,	2	2	0
8. Shock,	11	10	1
9. Embolism,	4	2	2
10. Causes not stated,	4	4	0
11. Persistent Vomiting,	1	1	0
	118	118	0
	137	137	0
II. Causes not primarily due to parturition—			
1. Pneumonia,	-	-	-
2. Phthisis,	-	-	-
3. Bronchitis,	-	-	-
4. Cardiac Disease,	-	-	-
5. Smallpox,	-	-	-
6. Scarlet Fever,	-	-	-
7. Lingering Disease,	-	-	-
8. Acute Gastritis,	-	-	-
	46	46	0
	51	51	0

We might take up the different causes of death and compare them during the different decades; but I shall only deal with two, viz. secondary postpartum haemorrhage and rupture of the uterus. Secondary postpartum haemorrhage occurred 7 times during the first decade with 5 deaths; during the second there were 4 cases, and during the third 3 without any deaths. This clearly shows a decided improvement in the conducting of the 3rd stage of labour, and also in the treatment of postpartum haemorrhage. The use of the hot douche has undoubtedly saved more lives than the cold one. There is a marked improvement in the results of the treatment of primary postpartum haemorrhage during the 3rd decade when the hot douche was freely used. As regards rupture of the uterus there were two in the 1st decade, and very probably a third one which is entered as a death from shock, but as the patient died a few minutes after version of a hydrocephalic child which forceps had failed to extract, I think rupture had likely occurred. All were fatal. In the 2nd and 3rd decades there were nine at least treated to a finish at their own homes. There was a case of spontaneous rupture in the 3rd decade which was transferred to the hospital and is included among the indoor cases.

In considering the operative work we learn that the forceps was applied nearly twice as frequently during the 2nd and 3rd decades as during the 1st. Craniotomy was done more frequently during the middle decade. The proportion of craniotomies is very small considering the large amount of rickets present in Glasgow. The explanation of the small number during the last ten years is due to the fact that such cases are usually transferred indoor. Married women used to object to come in, as the hospital in the olden days had a very bad reputation. Happily that is now becoming a thing of the past. The proportion of outdoor operative cases, taking them all round, is not a very large one. When we come to consider the operative work done indoors we shall find the very opposite to be the case.

The indoor cases are fewer in number, but the death-rate is exceedingly high. From the nature of the cases dealt with it could not be otherwise. I have already stated that a good

many of the most difficult cases are brought in from the outdoor department. Besides these we have many exceedingly bad cases sent in by private practitioners in Glasgow and its immediate neighbourhood, and also from the various towns in the West of Scotland. As a rule, these cases only reach us after they have been long in labour, and in many cases after repeated attempts have been made to deliver them. As illustrative of this take the 8 cases of ruptured uterus in the 3rd decade. Six were sent to us in a hopeless condition, in fact one died a few minutes after admission. Of the other two, one was a case of spontaneous rupture, already referred to as having occurred in the outdoor department, while the remaining one alone occurred in the hospital. This was a case of contracted pelvis delivered by antero-posterior forceps. The promontory perforated the back of the lower uterine segment. In the same way a good many cases of eclampsia and antepartum haemorrhage come to us practically moribund. By rights these deaths should not be entered, but it has always been the custom to do so. If we followed the method usually adopted in general hospitals of not counting such deaths our death-rate would be much diminished. In the 1st decade the patients were treated in the old hospital which stood on the same site as the present one. It was pulled down in 1879 and the present one put in its place. The old building was, I believe, unsuitable in many ways. The present one, which was opened in January, 1881, falls far short of the requirements of the present day. The labour rooms are far too small, there is no operating theatre, and no isolation ward for septic cases. I believe the old building was condemned because of so many of the cases becoming septic. If stone and lime were at fault the new building should have been free from this, but a study of the cases treated during 1881 shows the same series of high temperature, with occasional fatal cases of sepsis, as occurred before. This condition of matters continued more or less until in 1896 five cases of sepsis were removed to the fever hospital in one week, and there were many cases of high temperatures in the hospital. Overcrowding was generally blamed, and efforts were made to restrict the numbers admitted. Every antiseptic precaution was adopted during labour, and it

TABLE V.—SINGLE (INDOOR), 1869-78.

TABLE V.—SINGLE (INDOOR), 1869-78.

TABLE V.—SINGLE (INDOOR), 1869-78.

TABLE VII.—MARRIED (INDOOR), 1889-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	D.	A.	Total.
1	57	13	9	2	15	6	1		27	300	327
2	11	1	4	41	11		1		6	308	314
3	1	2	3	5	9	3	3		10	235	245
4		22	6		17				9	186	195
5		11	1		24	2	2		2	143	145
6		1		1	20	2	5		1	89	90
7		1	2	2	28	2	1		6	83	89
8		1	1		18	3	3		1	46	47
9					21	1	1		1	40	40
10					17	1	5		1	24	25
11					9	1	7		4	19	23
12				3	2	4	2		1	8	8
13					3	1	1		1	6	7
14				1	1	1	1		1	3	4
15					1	1			2	2	2
16						1			1	1	1
17					1				1	1	1
1	69	18	26	11	314	7	188	6	38	5	1494
									69		1563

Causes of death among the Indoor Cases from 1889 to 1898 :—

I. Primarily due to parturition—

	Married.	Single.	Total.
1. Septicaemia,	28	16	44
2. Primary Postpartum Haemorrhage,	1	0	1
3. Secondary "	0	0	0
4. Placenta Praevia,	5	1	6
5. Accidental Haemorrhage,	1	0	1
6. Eclampsia,	13	6	19
7. Rupture of the Uterus,	7	1	8
8. Shock,	1	2	3
9. Embolism,	0	0	0
10. Insanity,	0	1	1
11. Persistent Vomiting,	3	0	3
	59	27	86

II. Causes not primarily due to parturition—

	Married.	Single.	Total.
1. Pneumonia,	0	0	0
2. Phthisis,	0	0	0
3. Bronchitis,	0	1	1
4. Cardiac Disease,	2 (a)	0	2
5. Nephritis (Uræmia),	3	1	4
6. Pernicious Anaemia,	2 (b)	0	2
7. Delirium Tremens,	1	0	1
8. Exhaustion from Starvation,	1	0	1
9. Enteric,	1	1	2
	10	3	13

(a) Post-mortem Caesarean section was performed in one of these cases. The child breathed faintly, but did not survive.
 (b) Both of these died undelivered. Post-mortem Caesarean section was done on one. The child showed no signs of life.

MATERNITY HOSPITAL STATISTICS.

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	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	D.	A.	Total.	Total D.	GRAND TOTAL.
1	9	471	10	886	6	258	67	13	25	1697	1722	52	2049
2		34	3	415		243	61	14	3	772	775	9	1089
3			1	58		66	28	4	1	160	161	11	406
4						35	24	1		67	67	9	262
5						5	8	2	1	17	18	3	163
6							6	4		10	10	1	100
7							1	2		3	3	6	92
8							2			3	3	1	50
9								1					40
10												1	25
11												4	23
12													8
13												1	7
14												1	4
15													2
16													1
17													1
	9	505	14	1367	6	607	197	40	30	2729	2759	99	4322

Operative Cases, Indoor, 1889-98—										FATAL.	
										COMPLICATIONS.	
										FATAL.	
										FATAL.	
										FATAL.	
										FATAL.	
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TABLE VIII.—MARRIED (INDOOR), 1869-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	D.	A.	Total.
3	121 25 4	14 282 357 154 43 17 3 1	12 124 7 213 5 224 10 186 2 110 2 42 37 1	3 31 3 77 7 100 2 111 93 2 85 4 64 46 27 11 3 1 1 2	1 11 2 29 28 35 1 40 45 47 49 48 26 16 6 5 1 1 1	1 2 4 8 5 7 12 11 12 9 11 6 3 2 2 1	2 1 2 1			34 14 14 15 3 4 10 1 1 2 3 4 1 1 1 4 3 1 1	571 705 518 532 375 265 184 162 112 88 48 32 16 11 12 4 3 1 1	605 719 532 390 268 188 172 113 90 51 36 17 12 5 3 1 1
3	150	20	857	651	11	944	23			107	3096	3203

Causes of death among the Indoor Cases from 1869 to 1898 :—

I. Causes primarily due to parturition—

	Single.	Married.	TOTAL.
1. Septicaemia, - - - - -	54	41	95
2. Primary Postpartum Haemorrhage, - - - - -	1	3	4
3. Secondary " - - - - -	1	2	3
4. Placenta Praevia, - - - - -	2	8	10
5. Accidental Haemorrhage, - - - - -	1	1	2
6. Eclampsia, - - - - -	13	14	27
7. Rupture of the Uterus, - - - - -	2	11	13
8. Shock, - - - - -	6	8	14
9. Embolism, - - - - -	2	0	2
10. Insanity, - - - - -	2	0	2
11. Persistent Vomiting, - - - - -	0	3	3
12. Causes not stated, - - - - -	4	0	4
	-89	-89	-178

II. Causes not primarily due to parturition—

	Single.	Married.	TOTAL.
1. Pneumonia, - - - - -	4	3	7
2. Phthisis, - - - - -	5	2	7
3. Bronchitis, - - - - -	1	1	2
4. Cardiac Disease, - - - - -	2	2	4
5. Nephritis, - - - - -	4	4	8
6. Pernicious Anaemia, - - - - -	0	2	2
7. Delirium Tremens, - - - - -	0	1	1
8. Exhaustion from Starvation, - - - - -	0	1	1
9. Enteric, - - - - -	1	1	2
10. Cancer of the Cervix, - - - - -	0	1	1
	-19	-18	-35

TABLE VIII.—SINGLE (INDOOR), 1869-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	D.	A.	Total.	Total D.	Grand Total.
1	19	41	2184	9	146	4	27	81	4221	4302	115	4907
2	1	10	1061	2	173	7	43	20	2045	2065	34	2784
3		1	110		57	6	17	3	360	363	17	895
4			22		46	1	9		147	147	15	537
5			3		20		6	1	38	39	4	307
6				1	5	1	5	1	25	26	5	214
7					5	1	5		16	16	10	188
8					1	2	1		10	10	1	123
9					5		5		5	5	2	95
10					1		1		1	1	3	52
11											4	36
12					1				1	1	1	18
13											1	12
14											1	5
15					1				1	1		4
16												1
17												1
20	1285	52	3380	17	1591	12	467	106	6870	6976	213	10179

D

Operative Cases, Indoor, 1869-98—				Died.		COMPLICATIONS.		FATAL.
Forceps,	-	1025	(1 in 10)	27	(1 in 38)	Secondary Postpartum Haemorrhage,	5	2
Version,	-	214	(1 in 48)	26	(1 in 8)	Placenta Praevia,	63	10
Craniotomy,	-	152	(1 in 70)	36	(1 in 4)	Accidental Haemorrhage,	36	2
Induction of Labour,	90		(1 in 113)	7	(1 in 13)	Eclampsia,	74	27
Caesarean Section,	59		(1 in 172)	20	(1 in 3)	Rupture of Uterus,	13	13
Post-mortem "	2		(1 in 5080)	1	(1 in 1)	Occlusion of Os Uteri,	1	0
		—1554	(1 in 7)			Insanity,	14	1
						Scarlet Fever,	9	(?)
						Measles,	5	0
						Chorea,	3	0
						Enteric Fever,	3	2
						Malacosteon,	2	1
						Cancer of Cervix,	2	1
In considering the fatalities after operations, it must be borne in mind that not infrequently the death was not due to the operation. For instance, nearly all the eclampsia cases were delivered either by forceps, version, or craniotomy, and three of the fatal induction cases were done for uncontrollable vomiting.								FATAL.
COMPLICATIONS.								1
Primary Postpartum Haemorrhage,								119

In considering the fatalities after operations, it must be borne in mind that not infrequently the death was not due to the operation. For instance, nearly all the eclampsia cases were delivered either by forceps, version, or craniotomy, and three of the fatal induction cases were done for uncontrollable vomiting.

COMPLICATIONS.

Primary Postpartum Haemorrhage, 119

TAL.

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seemed quite evident to me when I took charge of the hospital in December, 1896, that the patients were being infected in the wards. Napkins to receive the lochial discharges were used over and over again. They were of course washed, and were supposed to be boiled. I had two of the supposed boiled napkins tested bacteriologically, and streptococci were found in both. My suspicions were confirmed and the explanation of the high temperatures made plain. A sterilizer was at once fitted up, and since then everything which comes in contact with the patients has been sterilized. Sterilized wool napkins are now used, and burned when soiled. The most rigid aseptic precautions are adopted during the labour and the puerperium. During or immediately after labour douching is never done except in operative cases, cases of postpartum haemorrhage, cases with a septic vaginal discharge, and when there is a macerated foetus. Vaginal examinations are made as seldom as possible, and only after the patient's external genitals have been thoroughly cleansed. The examiner's hands and forearms are rendered as aseptic as possible, and only lysol is used as a lubricant. All instruments are boiled immediately before being used. The result is that we now rarely have rises of temperature from septic absorption in the cases we have had to deal with from the first. If the case has been examined outside without the external genitals having been cleansed we often find a slight rise. We have had septic cases, but during the last $3\frac{1}{2}$ years we have only sent one septic case to the fever hospital, and she was septic on admission. Intrauterine douching during the puerperium is hardly ever necessary. In 1896 it was being done almost daily. As regards overcrowding, during last year we treated more patients in the hospital than in any previous year, and this year shows a marked increase over last.

We shall now examine the indoor cases.

	No. of Cases.	Total Fatal.	Approximate Proportion.
1st decade,	2,960	51	1 in 58
2nd ,,	2,897	63	1 in 46
3rd ,,	4,322	99	1 in 44
	<hr/> 10,179	<hr/> 213	<hr/> 1 in 48

This shews a marked rise in the death-rate.

If we take the deaths primarily due to parturition we find

	No. of Cases.	Fatal.	Approximate Proportion.
1st decade,	2,960	40	1 in 74
2nd ,,	2,897	52	1 in 56
3rd ,,	4,322	86	1 in 50
	<hr/> 10,179	<hr/> 178	1 in 57

This still shews a steady rise.

Of these 178 deaths, 95 were due to septicaemia, viz. :—

	No. of Cases.	Fatal.	Approximate Proportion.
1st decade,	2,960	20	1 in 148
2nd ,,	2,897	31	1 in 93
3rd ,,	4,322	44	1 in 99
	<hr/> 10,179	<hr/> 95	1 in 107

The proportion of deaths from sepsis as compared to other deaths primarily due to parturition is practically the same in the 1st and 3rd decades (viz. 50 and 51 per cent.), but it is slightly greater in the 2nd (60 per cent.). In the 1st decade the cases were treated in the old hospital, in the 2nd most of them were in the new building, and the remainder in a temporary hospital.

Taking the total cases the entire death-rate is 1 in 48; from causes primarily due to parturition (84 per cent. of the total deaths) 1 in 57, and from sepsis (53 per cent. of those primarily due to parturition) 1 in 107.

In considering the outdoor cases we saw that the death-rate has fallen markedly, while in the indoor cases we now see the very opposite. At first sight this does not seem very satisfactory, but there is a very simple explanation. A glance at the number of operative cases and complications dealt with will at once show the cause of the high death-rate. During the different decades the following operations were done :

	1st decade.	2nd decade.	3rd decade.
Forceps, - -	68 1 in 44	307 1 in 9	650 1 in 7
Version, - -	21 1 in 141	39 1 in 74	154 1 in 28
Craniotomy, - -	2 1 in 1480	38 1 in 76	112 1 in 39
Induction of labour,	2 1 in 1480	10 1 in 296	90 1 in 48
Cæsarean section,	...	3 1 in 987	56 1 in 77
,, post-mortem,	2 1 in 2161
Total, - -	<hr/> 93 1 in 32	<hr/> 397 1 in 7	<hr/> 1064 1 in 4

In the same way, if we compare the complications dealt with, a single glance will show the enormous difference in the cases. Take the cases of antepartum haemorrhage in each decade. In the 1st decade there were 2 cases of placenta praevia (both fatal) and 1 case of accidental haemorrhage. In the 2nd, 16 cases of placenta praevia (2 fatal), and 7 cases of accidental haemorrhage in a smaller number of cases. In the 3rd there were 45 cases of placenta praevia (6 fatal), and 28 of accidental haemorrhage (2 fatal).

In the 1st decade the cases were mostly normal. In fact the proportion of bad cases was much the same as in the outdoor department. During the last two decades more and more abnormal cases have been steadily coming to the hospital. The increase in the operative work has been enormous. The more frequent use of the forceps accounts for a good many of the operations. It goes without saying that the forceps is applied very much more frequently than formerly, and with the happiest results. No woman is now allowed to linger on in agony until she is exhausted. If with good pains the head is not making any progress after the 2nd stage has lasted two hours in a multipara or three in a primipara we make it a rule to deliver. A very large proportion of our forceps cases are high ones with contracted pelves, and in many delivery is only possible in the Walcher position. That the early application of forceps, and therefore their more frequent use, is beneficial to the patients is abundantly proved by the results in the different decades. In the 1st decade, of the 68 cases 12 died (1 in 6); in the 2nd, of the 307 cases 8 died (1 in 38); and in the 3rd, of the 650, 7 died (1 in 93). In the 2nd and 3rd decades some of these fatal forceps cases were really cases of antepartum haemorrhage and eclampsia, whereas in the 1st decade so few of these complications were dealt with that exceedingly few of the fatal forceps cases could have been of this nature. Sepsis shock and exhaustion were the causes of death in forceps cases in the 1st decade.

Versions and inductions of labour have also been much more frequent in the last two decades. Taking the major operations of craniotomy and Caesarean section we see a very great increase. It is very extraordinary that in the 1st decade

only two craniotomies were necessary. One of these was fatal. Very few cases of contracted pelvis were dealt with.

Deformity of the pelvis from rickets is so very common at the present day that we are practically never without one or two cases in the hospital, and have sometimes had as many as eight. Within the last two or three years fully 10 per cent. of the cases have had contracted pelvises, and by that I mean with a true conjugate of from $3\frac{3}{4}$ inches downwards. We have many others with very slight contractions. From the study of the records, it seems to me that rickets could not have been a very common disease among the infants of 50 years ago, but that after that it must have become much more common. At the present day we are dealing with the results of the disease of from 20 to 30 years ago. Sir Hector Cameron recently remarked to me that he was quite convinced that rickets was now on the decline in Glasgow. From a much more limited experience I had come to the same conclusion. Fifty years ago the city was not nearly so overcrowded, and I take it that the poor lived more on oatmeal, and what is of the greatest consequence to the infants, they were brought up on the breast, and not on condensed skim milk or starchy trash as at the present day. Loch Katrine water is of course generally blamed, but unjustly; I have seen just as bad cases of rickets in London as here, and yet the London water is just as hard as the Glasgow water is soft.

Fifty years ago sanitation, which is now doing so much in improving the public health, was in its infancy. In a few years I imagine the number of rickety pelvises will begin to diminish, but at the present time we are dealing with the results of the disease when it was at its acme.

The proportion of other complications is also very large. As I have already pointed out, many of these complicated cases are in a hopeless condition when admitted.

If we now compare the proportion of deaths among the married and single of the three decades we find:

	Married.	Total Fatal.	Primarily due to Parturition
1st decade,	810	11 1 in 73	9 1 in 90
2nd „	830	27 1 in 31	21 1 in 40
3rd „	1,563	69 1 in 23	59 1 in 27
Total,	3,203	107 1 in 30	89 1 in 36

TABLE IX.—MARRIED (OUTDOOR AND INDOOR), 1869-98.

	15-19		20-24		25-29		30-34		35-39		40-44		45-49		50-54		55-59		D.	A.	Total.
1	13	1641	31	3184	17	785	7	192	2	71	1	18	4			1			71	5895	5966
2	1	359	7	3720	13	1599	6	433	3	132		27	1						30	6272	6302
3		43	5	2179	12	2442	10	743		206		38	1						27	5652	5679
4		5	4	902	13	2553	6	1111	1	378		48							24	4998	5022
5			1	232	9	1909	3	1425	4	474	2	76	6						19	4142	4161
6				53	6	1092	7	1594	4	690		127	10						18	3567	3585
7				17	2	488	10	1415	4	851	7	156	9						23	2937	2960
8				5	2	186	6	969	3	912	2	227	16						13	2317	2330
9				1		66	2	555	8	862	2	253	15						11	1754	1765
10						15	1	257	4	600	3	273	22						8	1169	1177
11					1	9	1	86	9	346	5	274	21						18	738	756
12						6		27	2	175	2	193	29						4	430	434
13						1	1	18	2	100	1	97	5						4	221	225
14						1		4		29	1	51	11			2	1		1	99	100
15								2		11		26	4							43	43
16										5		8	4							17	17
17										1		6	1							8	8
18										1			1							7	7
19										2		5	1							3	3
20													1							1	1
	14	2048	48	10,313	75	11,152	60	8831	46	5846	26	1903	2	163		13	1	271	40,270	40,541	

Causes of death among the Outdoor and Indoor Cases from 1869 to 1898:—

I. Causes primarily due to parturition—

	Married.	Single.	TOTAL.
1. Septicaemia, - - - - -	79	62	141
2. Primary Postpartum Haemorrhage, - - - - -	19	2	21
3. Secondary " - - - - -	7	1	8
4. Placenta Praevia, - - - - -	25	2	27
5. Accidental Haemorrhage, - - - - -	10	1	11
6. Eclampsia, - - - - -	23	19	47
7. Rupture of the Uterus, - - - - -	13	2	15
8. Shock, - - - - -	16	9	25
9. Embolism, - - - - -	2	4	6
10. Insanity, - - - - -	0	2	2
11. Persistent Vomiting, - - - - -	4	0	4
12. Causes not stated, - - - - -	4	4	8
	207	108	315

II. Causes not primarily due to parturition—

	Married.	Single.	TOTAL.
1. Pneumonia, - - - - -	15	7	22
2. Phthisis, - - - - -	13	7	20
3. Bronchitis, - - - - -	10	1	11
4. Cardiac Disease, - - - - -	8	2	10
5. Smallpox, - - - - -	1	0	1
6. Scarlet Fever, - - - - -	1	0	1
7. Lingering Disease, - - - - -	4	0	4
8. Acute Gastritis, - - - - -	2	2	4
9. Nephritis, - - - - -	4	4	8
10. Pernicious Anaemia, - - - - -	2	0	2
11. Delirium Tremens, - - - - -	1	0	1
12. Exhaustion from Starvation, - - - - -	1	0	1
13. Enteric, - - - - -	1	1	2
14. Causes of the Cervix, - - - - -	1	0	1

TABLE IX.—SINGLE (OUTDOOR AND INDOOR), 1869-98.

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	D.	A.	Total.	Total D.	Grand Total.	
1	27	2362	50	3322	9	870	10	199	4	42	8	100	6806	12,872
2	1	129	13	1494	8	864	2	230		53	8	24	2778	9104
3		2		201	2	260		88		27	7	3	585	6267
4				50		115		67	1	17	3	24	252	5274
5				7		29		36		11	1	1	84	4246
6				1		16	1	22		14	1	19	55	3641
7						1		16		11	2	23	30	2990
8						1		2	1	5	2	14	15	2346
9								7		7	9	11	9	1774
10								2		7	2	8	2	1179
11								1		1	1	18	1	757
12										1	1	4	1	435
13												4		225
14												4		100
15											1	1		44
16										1				17
17														8
18														7
19														3
20														1
	28	2493	74	5075	19	2156	13	668	5	191	32	130	10,619	51,290
												401	10,749	

Operative Cases, Outdoor and Indoor, 1869-98—			FATAL.
Forceps, - - - - -	(1 in 21)	Rupture of the Uterus, - - - - -	15
Version, - - - - -	(1 in 98)	Occlusion of Os Uteri, - - - - -	0
Craniotomy, - - - - -	(1 in 267)	Inversion of Uterus, - - - - -	3
Induction of Labour, - - - - -	(1 in 537)	Insanity, - - - - -	1
Cæsarean Section, - - - - -	(1 in 870)	Smallpox, - - - - -	1
Post-mortem - - - - -	(1 in 25,645)	Scarlet Fever, - - - - -	1 (?)
	FATAL.	Typhus, - - - - -	2
Primary Postpartum Haemorrhage, - - - - -	405	Enteric, - - - - -	3
Secondary - - - - -	19	Measles, - - - - -	5
Placenta Praevia, - - - - -	168	Cancer of Cervix, - - - - -	3
Accidental Haemorrhage, - - - - -	184	Chorea, - - - - -	5
Eclampsia, - - - - -	139	Malacosteon, - - - - -	2
	COMPLICATIONS.		

	Single.	Total Fatal.	Primarily due to Parturition.
1st decade,	2,150	40 1 in 54	31 1 in 69
2nd „	2,067	36 1 in 57	31 1 in 67
3rd „	2,759	30 1 in 92	27 1 in 102
Total,	6,986	106 1 in 66	89 1 in 78

It is rather curious that, except in the 1st decade, the death-rate among the married is much the higher. Taking the total in the three decades, the death-rate among the married is fully double that among the single. This is contrary to what one would expect, but the explanation will be found in the fact that the vast majority of patients sent in by practitioners are married. They are all very difficult cases and some of them hopeless.

Many interesting details might be brought out by comparing the results of treatment of the complicated cases in each decade, but space will not permit me to do this.

In Table IX. we have the whole of the cases combined.

		Total Fatal.	Primarily due to Parturition.
Married,	40,541	271 1 in 150	207 1 in 196
Single,	10,749	130 1 in 83	108 1 in 100
Total,	51,290	401 1 in 128	315 1 in 164

The death-rate among the single cases all over is thus seen to be much higher than among the married. The death-rate among the primiparae, especially among the single ones, is much higher than with subsequent births until we reach the 11th among the married, when it reaches its highest point (1 in 42).

In the near future I hope to publish an elaborate series of tables giving the percentages of deaths according to age and number of the confinement. Mr. Gunn, of the Scottish Amicable Life Assurance Society, to whom I am deeply indebted, has kindly drawn up the tables for me.

ECLAMPSIA: AN ANALYSIS OF THE CASES WHICH HAVE OCCURRED IN THE GLASGOW MATERNITY HOSPITAL DURING THE LAST FIFTEEN YEARS.

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IN the present paper¹ we give an analysis of the cases of eclampsia which have occurred in the Glasgow Maternity Hospital during the last fifteen years. They are eighty cases in all. We have not referred to those that have occurred in the outdoor practice of the hospital, as details of them are very meagre, and often indeed entirely wanting.

We desire to say that there has been no attempt made to consider the subject of eclampsia exhaustively. We have only pointed out the results of our examination into different questions connected with the disease, and compared our conclusions with those of other writers who have approached the subject in a similar way.

Number of Births, Age, Plural Pregnancies, Recurrence of Eclampsia in the same Subject.

Like all others who have examined a long series of cases, we have found that primiparae are much more frequently attacked than multiparae. The percentage of primiparae in our list is 72. Others give very much the same figures—Dührssen 84 per cent., Olshausen 74 per cent., Bidder 74 per cent., Knapp

¹ I am indebted to James Husband, M.B., C.M., late House Surgeon to the Hospital, for his assistance in preparing the table at p. 80.

73 per cent. As regards age we cannot say that old primiparae are more frequently affected than young. In the 53 primiparae 11 were over 25 years, and the average age was 22. In the 20 multiparae the average age was 28. Knapp found the average 24 and 29 respectively. Both Winckel and Bidder found old primiparae only slightly more liable to eclampsia.

The frequency of plural pregnancy apart from eclampsia is variously stated. Olshausen puts it at $1\frac{1}{4}$ per cent., Bidder 2.1 per cent., Winckel 1.1 per cent. The same authorities found the frequency in eclampsia 8 per cent., 7.4 per cent., 11 per cent. respectively, and Dührssen found it 4.5 per cent., Zweifel 6 per cent. Our results give a frequency of 3.7 per cent.

The recurrence of eclampsia in the same subject is extremely rare. Olshausen, for example, has only met with it thrice, and Dührssen also mentions only three cases. In our list there are two cases mentioned, Cases 11 and 13.¹ The former, a V.-para, who died, had convulsions at her three previous pregnancies; and the latter, a V.-para, had convulsions in all her previous labours. In both the urine became solid on boiling. These cases are comparable with one mentioned by Lumpe, in which eclampsia occurred in the 1st, 2nd, and 5th labours.

The Time of Onset and Course of the Disease.

In eclampsia it is very striking the large number of pregnancies that terminate prematurely. 50 per cent. of the cases we have tabulated, and in which the time of onset is stated, occurred before "full time." This is in agreement with the tables of other writers. Olshausen's cases show 30 and 40 per cent., Bidder's 30 per cent., Green's 33 per cent.,

¹In private practice I have seen only one case, a II.-para, who had eclampsia in a previous pregnancy. She was the subject of chronic nephritis. In the second pregnancy, about the 7th month, several convulsions occurred, and I saw her in consultation with Dr. Campbell, of Govan. In that case I dilated the os forcibly with my fingers—it was not rigid—turned and extracted the child. The operation took about $1\frac{1}{2}$ hours. We then transfused two pints saline solution and gave morphia hypodermically. Patient made a good recovery.

Faustel's 37 per cent. The earliest time of onset in our cases was at the 6th month. In Bidder's cases the earliest was in the 5th month, in Green's at 21 weeks, but Olshausen has seen cases in the 3rd and 4th months, and Norris one in the 4th month.

Another point of interest in connection with the time of onset is the relative frequency as regards pregnancy, labour, and puerperium. The following are the figures given by some recent writers :

		Ante-Partum.	Intra-Partum.	Post-Partum.
Bidder	(455 cases),	8·3 per cent.	64 per cent.	27 per cent.
Dührssen	(200 „),	27·5 „	48·5 „	24 „
Goldberg	(81 „),	25 „	57 „	14 „
Green	(36 „),	36 „	22 „	42 „
Herman	(12 „),	75 „	16 „	8 „
Knapp	(22 „),	9 „	68 „	22 „
Moran	(71 „),	49 „	31 „	19·5 „
Olshausen	(200 „),	— „	— „	14 „
Schroeder	(316 „),	19 „	60 „	20 „
Weissheimer	(21 „),	9·5 „	71·4 „	19 „

The proportion of cases in which the seizures begin post-partum is readily determined, and, roughly, may be said to be somewhere about 20 per cent. In our cases it was only 6 per cent. Green's 42 per cent. is exceptionally large. It is frequently very difficult, however, to determine the relative frequency of intra and ante-partum eclampsia, for it is often a question whether the eclampsia has induced the labour, or the uterine contractions of labour have been the exciting cause of the convulsions. Besides being of interest in studying the disease in the abstract, the deciding whether labour has or has not commenced may be of importance in particular cases, as it may influence the treatment. This will be seen when that part of the subject is under consideration. Of our 72 cases, where an exact statement of the onset of the convulsions is given, we find in 30 (40 per cent.) it occurred ante-partum, in 22 (30 per cent.) intra-partum, and in 16 cases uncertainty existed whether it was ante or intra-partum.

When convulsions occur during pregnancy, labour as a rule soon comes on. It is generally stated that the

disease rarely quiets down under treatment and allows the pregnancy to continue. Among the 80 cases tabulated, only in Case 61 did the woman leave the hospital undelivered. It happened, however, that in Case 41 labour did not occur till a month, in Case 52 not until seven days, in Case 74 not until three days, and in Case 78 not until two days after the convulsions had ceased and the disease had been arrested. Many other writers have mentioned cases cured during pregnancy. Olshausen had five in his 200 cases, Green had two, Sloan mentions one, and Moran describes a most interesting case where the woman was semi-comatose after several fits, and where the disease was arrested and labour did not occur until two months later.

Cases 7 and 41 are of special interest, because of the long intervals between the convulsions; the former had the five convulsions spread over as many days, and the latter one fit on the day of outbreak and five fits just before delivery, two days later. Olshausen mentions a case where the woman had 31 fits spread over seven days: she recovered.

The death of the foetus, as first suggested by Winckel, is stated by many to be the cause of the arrest. We are inclined to share this belief to some extent, for many of the children are born dead after arrest of the seizures—witness Cases 52 and 74. From our arrested cases, and those of other writers quoted, we can only say that nearly all the children were born dead; and that although a macerated foetus is sometimes born at the time of the convulsions—witness Cases 63 and 78—still the death of the foetus does to some slight extent probably control the seizures.

There is one point in connection with this matter of arrest—it is that in arrested cases convulsions very rarely recur at the time of labour. Bidder, Olshausen, and Schroeder all refer to this.

Speaking generally, when the convulsions begin during labour they do so early or late in that process.

As regards the puerperal cases, the seizures usually commence shortly after delivery, although we had one case where they did not come on until the fifth day.

Prodromal Symptoms and Condition of Urine.

The prodromal symptoms of eclampsia are oedema, headache, vomitings, epigastric pain, disturbances of vision, albuminuria. Of the cases where the prodromal symptoms are referred to, in only six is it stated that there were none.

The most constant was oedema, but it was extreme in only Cases 9, 24, 38, 76. It lasted for days, weeks, and often months before the seizures occurred. Weissheimer and Goldberg both found oedema in 50 per cent., and Olshausen and others found it in the great majority of their cases.

Of greater importance than oedema as premonitory symptoms are headache, epigastric pain, disturbance of vision, etc.

Headache, which is very frequently present prior to the attack—we found it noted in 33 cases—may continue for long before the seizures; in Cases 4 and 5 for instance it was present for months. When severe, however, and especially if it comes on suddenly and is associated with any of the other premonitory symptoms, it indicates that the convulsions are not far off; for example Cases 24 and 52. It is usually frontal in situation, but in two cases it is mentioned as being occipital.

Sickness and vomiting are very generally complained of. Epigastric pains, however, which so many mention, is only noted in Cases 38, 39, 42. Our figures in this respect agree with Dührssen's, who only found it mentioned once, rather than with those of Olshausen and others, who consider the symptoms fairly general.

Disturbances of vision if extreme indicate the near approach of convulsions—witness such cases as 9, 13, 54. Minor degrees of such disturbances, however, may continue for long; in Case 7 they continued for a week, and in 56 for a month before the fits came on.

Other symptoms are of little importance—pain in the back, which one might expect and which some represent as being frequently present, we have only found mentioned in one case.

In no case was there any aura preceding the convulsions. Olshausen, however, has observed it in one or two cases.

The condition of the urine is unfortunately often not so carefully stated as one would desire. When there is a state-

ment regarding it, albumen was always found, and in only four cases (16, 33, 67, 70) is the amount stated to have been small. Olshausen's results were very much the same. Among 168 cases only once was albumen entirely absent, and only in four cases present in small amount. Winckel found albumen present in 84 per cent., Green in 100 per cent., and Dührssen in 99 per cent. of their cases. The quantity of albumen, however, varied greatly in our cases. It is usually stated to have been "distinct," "abundant," "solid on boiling," etc. As regards the tube casts and blood, we find in 17 cases the former, and in 8 the latter, but we are sure that these figures in no way represent the frequency of these abnormal constituents. Green for example reports casts and blood being present in 30 out of 36 cases, while Knapp found them in 72 per cent., and Dührssen in 60 per cent. of their cases.

Mortality and Prognosis for the Mother.

Out of our 80 cases there were 30 deaths, which gives a mortality of 37·5 per cent. It is an appalling death-rate, and compares unfavourably with the figures given by most other writers. Take for example the following: Olshausen 25 per cent., Green 25 per cent., Goldberg, 24·7 per cent., Zweifel 22 per cent., Dührssen 21 per cent., Schroeder 20 per cent., Bidder 17·3 per cent., Fehling 17 per cent. The only figures at all comparable to those given by us are such old figures as Hoffman's 39 per cent., Bailly's 42 per cent.

Winckel's mortality of 7 per cent. with chloral treatment is most surprising, but even it is eclipsed by G. Veit's 3 per cent. in over 60 cases with morphia treatment. Knapp in 22 consecutive cases reported by him had only one death. His cases also were treated with morphia.¹

Of the 30 fatal cases, two died undelivered. In 11 cases an autopsy was permitted, and the results were as follows:—The kidneys were found diseased in seven cases, the liver in three, the lungs in two, while one died of septic peritonitis, and one of a ruptured duodenal ulcer on the sixth day of the puerperium.

¹ Stroganoff reported the other day 58 cases with one death, a mortality of only 1·7 per cent.

Regarding the other fatal cases it is useless to speculate.

A very striking feature of two of the cases (73 and 76) was a pronounced degree of flatulent distension of the abdomen; so great indeed was it that the question of septic infection was raised; but there was no other evidence of such a condition ante-mortem, and none at the autopsy.

We have not gone into any details regarding the conditions found at the autopsies, because the reports are so ragged and incomplete.

As influencing the maternal mortality, and consequently the prognosis, we would mention the "parity," the time of most of the fits, the frequency, severity, and number of the fits, the condition of the urine as regards quantity and albumen, the condition of the pulse and the temperature, and the treatment that is adopted.

As regards "parity" we can only say that our figures are quite contrary to the frequently expressed opinion that the disease is more fatal amongst multiparae than amongst primiparae. Our figures and those of others are the following:

Cases in Table,		Primiparae.		Multiparae.	
		42 per cent. fatal.		23 per cent. fatal.	
Dührssen,	.	19	" "	28	" "
Goldberg,	.	21	" "	45	" "

Olshausen found the mortality the same in both. Taking it all over the prognosis, then, is rather less favourable for multiparae than for primiparae.

The Influence of the Convulsion.

(a) *Time of onset of the seizures.*—Our mortality, as we have said, was 37·5 per cent. over all. In the ante-partum cases it was 43 per cent., in the intra-partum cases 28 per cent., and in the post-partum cases 0 per cent. The following are the results of recent writers:

	Pregnancy.	Labour.	Puerperium.
Dührssen, .	38 per cent.	19 per cent.	12 per cent.
Goldberg, .	65 "	11 "	0 "
Green, .	46 "	25 "	6 "
Olshausen, .	25 "	25 "	25 "
Weissheimer,	50 "	26 "	0 "
Zweifel, .	17 "	14 "	10 "

It is perfectly evident, therefore, from the above figures that the prognosis is most grave when the seizures begin during pregnancy, and least grave when they occur during the puerperium. Olshausen is the only one who takes exception to this opinion, and he does so on the ground that in his 200 cases he had a mortality of 25 per cent. in pregnancy, labour, and puerperal cases alike. When the disease breaks out in the puerperium, he considers that if the fits occur late (12 hours after birth), the prognosis is very much graver than if they occur early. One of our cases (No. 28) had the first seizure on the 5th day, and the disease ran a very mild course.

The earlier the onset is in labour and pregnancy the graver is the prognosis. In our labour cases, when the seizure began in the second stage we had 1 death in 5 cases (20 per cent.); but when the fits began in the first stage we had 5 in 16 (31 per cent.). In our cases, when the fits came on in pregnancy before the seventh month we had 6, with a mortality of 3 (50 per cent.), in those that occurred in the seventh and eighth months 23, with 11 deaths (43 per cent.), while in those at full time we had 35, with 13 deaths (37 per cent.).

(b) *Number, frequency, and severity of fits.*—Speaking generally, the more numerous the fits the graver is the outlook. In the cases that proved fatal, the average number of fits was 10·6, and in those that recovered it was 5. Green found that in the cases that recovered the average number was 5·3, and in those who died 10·8. To look at it in another way, Bidder found that in cases where there were more than 16 fits there was a mortality of 43 per cent., while in those with less than 16 it was only 7·5. In our cases we find that when there were 12 fits or more the mortality was 55 per cent., and when there were less it was only 31 per cent.

But there are many exceptions to the general rule above expressed. Thus, Olshausen mentions a case with 39 fits, and Ahlfeld one with 82, both of which recovered. Winckel says he has never seen a case with more than 20 fits recover. In our list are two cases which had 21 and 28 fits respectively, and yet recovered.

On the other hand, it must be remembered that some fatal cases have very few fits; for example, amongst our list two

had two fits (Cases 6 and 21), and one had only one (Case 29). Such cases, however, are not very common; they are usually associated with early coma, and often with very inveterate and complete suppression of urine—witness Case 29.

Much more important than the number are the frequency and severity of the fits and the degree of coma present. Regarding these points, however, we unfortunately can give no statistics. We can only join with all other writers in saying that the more frequent and severe the fits, and the earlier and deeper the coma, the graver is the prognosis.

The Urine and the Prognosis.

As regards the urine the principal factor influencing the prognosis is the quantity passed. Prior to the seizures there is frequently a diminution in the quantity, and in one or two of our cases there was such a history. In two others, however, the reverse was the case, the women stating that they had been passing larger quantities. Mere general statements, however, are of little value in this connection, as an increased frequency of micturition is often considered by women synonymous with an increased quantity passed, often quite an erroneous conclusion. Herman, who made very careful examinations of the urine in twelve cases, found the quantity of urine diminished during the fits in nine cases and increased during the fits in two.

Speaking generally, the outlook is graver when the quantity of urine passed is small, and particularly is this the case after delivery and when the kidneys do not respond to fluids administered either by the mouth or by transfusion. When there is suppression of urine the prognosis is absolutely bad.

The quantity of albumen present to some slight extent influences the prognosis, although we cannot say that the smaller the quantity the milder will be the disease. Indeed, in three of our cases (16, 67, 70), when the quantity is stated to have been small, the subjects had 12, 14, and 15 fits respectively. Of Olshausen's four cases, when the quantity of albumen was small, three ran a mild course, but one had 23 fits.

Our tables show that, with very few exceptions, the quantity

of albumen was large, and Herman in connection with his twelve cases makes the remark, "In all the cases there was a time in the course of the case at which the urine was solid or nearly so with albumen." That statement of Herman, and our own experience, leads us to make the remark that the albumen comes and goes very quickly in the course of this disease. It is surprising how quickly it disappears after delivery, as every one knows, but it is equally surprising how suddenly it sometimes appears and increases.

Influence of Temperature and Pulse on the Prognosis.

As regards the influence of the temperature on the prognosis, we can offer no opinion. We would only quote Herman, who, from his careful investigation of the subject in twelve cases, says: "These cases do not show that the fits have any immediate or direct effect on the temperature."

Unfortunately, regarding the pulse and its influence in the prognosis, we can only generalise, for there is no very careful record of it except in a few cases. The pulse we have always found full and bounding at first, and always quicker after a fit; also that it becomes quicker, more compressible, and, later, intermittent as the fits continue and the coma becomes more profound. While great frequency, compressibility, and intermission are extremely unfavourable conditions, they do not make the case quite hopeless, for the pulse in Nos. 66 and 78 had all these features, and yet after a saline injection it steadily improved, and the women made an excellent recovery.

Mortality and Prognosis for Child.

For the child the prognosis is very bad. When it has reached a viable age the mortality is 57 per cent. This may be compared with the following:

Winckel,	. 77 per cent.	Zweifel,	. 34 per cent.
Green, .	. 69 ,,	Olshausen,	. 28 ,,
Dührssen,	. 49 ,,	Bidder,	. 23 ,,
Goldberg,	. 47 ,,	Weissheimer,	20 ,,

The foetal mortality is lowered when we consider that in 7 cases craniotomy was performed, sometimes on account of contracted pelvis, but more frequently simply to facilitate the extraction of the child. In only two cases had the child reached "full time," and in some of the cases it had not even reached a "viable" age.

The principal circumstances influencing the prognosis are the age of the foetus, the number of seizures before its birth, the treatment adopted to bring about its delivery and avert the disease. As regards the age of the foetus, we find a mortality in those born at the seventh month of 70 per cent., in the eighth of 62 per cent., and in the ninth of 49 per cent.

The effect of the convulsions on the foetus may be seen by the fact that where there were ten or more fits, only two children were born alive. In these two cases one mother had 12 and the other 14 fits. Olshausen in his 200 cases had 13 children born alive where there were more than 15 seizures. Indeed, two mothers had 26 fits, spread, however, over a considerable time.

But even when the children are born alive, not a few die shortly after birth. On our list we find two cases (Nos. 6 and 42), where the child died of convulsions, the one on the fourth and the other on the tenth day.

This occasional occurrence of convulsions in the child after delivery has been remarked on by most writers. Only the other day Fitzgerald reported a case where the child had 39 fits during the first few days of its life. It ultimately recovered and remained perfectly well. Several observers have found albumen present in such infants' urine. Case 65 is an example of this.

As regards treatment, which next to age has probably the greatest influence on the foetal mortality, we can only say that since the introduction of saline transfusion the death-rate has fallen very markedly. Morphia by many is said to act injuriously on the child, and one or two children have been born with symptoms of poisoning. Chloral we are not aware has any bad effects on the child. The great advocate of chloral, Winckel, gives, however, his foetal mortality as 77 per cent., but that is due to his great objection to operative interference.

The two things to attend to when the child is being considered are, to arrest the fits, and to evacuate the uterus as quickly as possible.

TREATMENT.

There is probably no subject in obstetrics regarding which greater differences of opinion exist than regarding the treatment of eclampsia.

Certain questions may, however, be considered settled. For instance, every one is agreed on the importance of prophylaxis, that pilocarpine is a most dangerous drug in the acute stage, and that chloroform, although it quiets the restlessness and fits, is dangerous if administered for hours on end, as was the custom at one time. But more important than these is the admission, by all who have tried the treatment, that saline solution transfused directly into a vein or into the cellular tissue is a most valuable diuretic. For details regarding this method of treatment which Porak first introduced, and which in this country has been specially advocated by Jardine, we refer the readers of this paper to a contribution by the latter author in the *British Medical Journal* of May 26th, 1900.

The solution now used in the Maternity Hospital contains a drachm of chloride of soda and the same of acetate of soda to the pint of boiled water at the temperature of about 104°. One or two pints are transfused into the loose cellular tissue beneath the breasts.

There is not the least doubt that both the maternal and foetal mortality in the Glasgow Maternity Hospital have very appreciably fallen since transfusion of salt solution has, by general consent of the staff, become a routine part of the treatment. The maternal mortality before the saline transfusion was introduced by Jardine was 44 per cent., while since that time it has been 24 per cent.¹

The hot pack and steam baths have also sometimes acted beneficially.

¹ Case No. 63 must be excluded, as she died of a ruptured duodenal ulcer on the 6th day.

The two great matters of discussion as regards the treatment of eclampsia are: (a) The most suitable and effective drug to administer for the arresting of the fits; (b) when and how the uterus should be emptied.

The Medical Treatment.

The drugs that come under consideration, in what may be termed the medical treatment, are chloral, morphia, and veratrum viride. Examination of the table, we are sorry to say, does not furnish us with as much information and guidance as we would desire. Many of the cases were treated by more than one of these drugs, and some by all three, so that we could not say which, or if any, of the drugs was successful in relieving the condition. And this is the more to be regretted, as the cases where a vacillating line of treatment has been pursued have usually been severe and often fatal.

There have been, however, 49 cases, in each of which one only of these drugs was used.

Veratrum viride.—On looking over the table you will find that this drug was used very little before the saline transfusion came into vogue. At odd times it was used, as can be seen, but more usually in association with chloral: indeed there were only two cases where the drug was used alone, and the results were one death and one recovery. Since the saline transfusion was introduced there have been 7 cases with one death, a mortality of only 14 per cent. Such a result appears very satisfactory, but it must be mentioned that in two cases the drug failed to relieve the patient's condition, while chloral with or without potassium bromide did. The drug, early in the disease, when the pulse is full, bounding, and rapid, always lowers its tension and rate, and was administered often, it was thought, with benefit. When, however, the convulsions and coma had been present for some time, as is usually the case in the patients who are sent to the hospital, the pulse becomes soft and rapid, and veratrum viride can hardly do good, and sometimes, we have thought, has done harm.

America supplies the largest number of advocates of this drug. The most favourable statements regarding it are by Hirst, who said that in 9 years in the Maternity Hospital he had only had 2 deaths, and Parvin, who said at the Geneva Congress in 1896, that about 92 per cent. recovered. Both Norris and Jewett strongly recommended the drug. The latter author says: "From ten to twenty minims of the fluid extract of veratrum viride given subcutaneously should as a rule be the initial dose. Ten minims more may be given in the same manner every half hour till the pulse remains below sixty to the minute" (p. 527).

In this country odd cases of treatment with this drug have been reported, but no series of cases as far as we can discover.

The latest figures are these by Mangiagalli, who reported 18 cases with 17 recoveries.

Morphia.—The present revival of morphia and opium in the treatment of eclampsia arose from a paper by Prof. G. Veit, which appeared in the *Sammlung Klinische Vorträge* in 1888. In that paper the author stated that in 60 cases he had only 2 deaths, or a mortality of 3·3 per cent. The German school, with few exceptions, have been very strong advocates of the drug. Bidder, whose mortality was only 17 per cent. in 400 cases, speaks most highly of it, as do also Knapp and Fehling, with 4·5 (22 cases) and 17 per cent. mortalities respectively.

In our 8 cases treated by morphia, since the saline transfusion was introduced, 3 died, which gives a mortality of 37 per cent. The large doses that Veit recommends have never been given. He advises $\frac{1}{2}$ gr. as the first dose, to be followed by a $\frac{1}{4}$ gr. every two hours until the fits are arrested. Not more, however, than 2 grs. are to be administered in 24 hours.

The strongest advocates of the morphia treatment in this country are in the Rotunda Hospital, Dublin. In the last there years (1893-96) there were 8 cases with 2 deaths, a mortality of 25 per cent., the same as Olshausen, while in the first three years (from 1889-92), with chloral it was 6 and 17 and 35 per cent. (Table B in Jellett's text-book). We have not mentioned year 1892-93, because, although there were 9 cases and 2 deaths with chloral, morphia may

have been used in some of the successful cases. If that was so it will improve the case for morphia, and make it a little worse for chloral.

In Germany the treatment by morphia is very much favoured. There seems with many, however, a tendency to administer the drug in smaller doses than Veit recommends. Fehling quite recently, for example, while supporting the treatment by morphia, objected to large doses, as he had seen symptoms of poisoning. Bidder and others have remarked this also. There is no doubt that occasionally the infant is distinctly affected by the drug, and it would seem has once or twice died from it.

Chloral.—The great advocate of chloral, and the one who has had the best results, is Winckel. In his text-book on Midwifery he mentions having had only 7 deaths in 92 cases. Few in Germany, but many in France and this country favour it. Indeed in England it is still the favourite drug for combating the convulsions.

Of our cases treated by chloral, since saline transfusion became general, the table shows a mortality of 20 per cent., while prior to that it was 38 per cent.

In the great discussion on eclampsia that took place at the Geneva Congress in 1896, Charles, Charpentier, and Tarnier all spoke in favour of the drug, while Byers and Veit advocated morphia.

At the present moment it is impossible to say which of these drugs is the best, because brilliant results have been obtained with each of them. The striking feature about this matter however is, and we do not think it has been drawn attention to before, the most brilliant successes with each of the drugs have been obtained when each has been given boldly. Is it not possible that there the secret in great part lies?

Before leaving this subject of the medical treatment of eclampsia, we desire to draw attention to a paper by Prof. Stroganoff which appeared in the October number of the *Monatsschrift für Geburtshülfe und Gynäcologie*. The author reports there 58 cases without a death. One case, however, died of pneumonia, and we cannot quite agree with the author

in excluding it. But even reckoning it as a death from eclampsia, we get the exceedingly low mortality of 1·7 per cent. in 58 cases. Both morphia and chloral were used in the treatment of the cases. Stroganoff recommends in severe cases the giving of three hypodermic injections of morph. mur. ·015 grms., then administering per rectum 1·5-3·0 grms. chloral hydrat. per dose from 2-4 hours after the last morphia injection. Only if a convulsion is very threatening is morphia again to be used.

The Obstetrical Treatment.

As regards the obstetrical treatment of eclampsia, there is almost as great diversity of opinion as we have seen exists regarding the medical. While some few authorities, such as Winckel and Charpentier, caution against operative interference except of the very simplest nature, such as applying forceps at the outlet when the os is fully dilated, others, as Dührssen, go to the opposite extreme and advise emptying the uterus with all speed by making free incisions into the cervix, and if need be into the vagina and perinaeum. The latter are very few, and the former, except in France, are not numerous. The most of writers recommend a middle course, and favour emptying the uterus cautiously and when the indications for doing so are pressing.

It is at once apparent that doubt regarding the course to pursue can only exist, except amongst the few who would never interfere, in cases where the convulsions come on during pregnancy or early in labour; as when they commence during the second stage, or late in the first, all will admit that the clear indication is to empty the uterus either by forceps or traction on the limbs.

It is therefore where the disease has to be dealt with during pregnancy or very early in labour that the great differences of opinion exist regarding the treatment.

The great argument in favour of emptying the uterus is that, in a large majority of the cases, with the evacuation of the uterus, the convulsions cease or are greatly lessened in severity and frequency. Let us see what light our cases and those of others throw on this matter.

Of 18 cases where there is an exact record of the fits, and where the onset of the convulsions occurred during pregnancy, 13 had no fits after delivery ; and of 14 cases where it was doubtful whether the convulsions had come on during pregnancy or just at the commencement of labour, 10 had no fits after delivery. That is to say, in 71 per cent. of the cases such as we are considering there were no fits after the uterus was emptied. All other cases except one were treated actively, that is to say, the cervix was cautiously dilated, and labour was induced and the child extracted.

It must be mentioned, however, that of 5 cases where the disease occurred in the second stage, and where extraction with forceps was as speedily performed as possible, in only one was there no convulsions after delivery.¹

Turning to other writers we find they give the following results. Olshausen found in 64 per cent. the fits did not recur after delivery ; Bidder found expulsion of the child had a favourable effect in at least half of the cases, and in 33 per cent. there were no fits at all ; Dührssen writes that in 89 per cent. of his cases the convulsions ceased with evacuation of the uterus ; and Zweifel found that in 65 per cent. the fits entirely ceased and only one occurred after delivery.

All these results would seem to indicate that the uterus should be evacuated as soon as possible.

The subject, however, will be best discussed by considering separately the treatment of the two varieties, eclampsia in pregnancy and eclampsia in labour.

When the seizures begin early in labour, the cervix can usually be dilated, first with the fingers and then with "bags," and this is the treatment most generally recommended. Here we would remark in passing, it must be remembered in carrying out this, as indeed any other operative treatment in eclampsia, the woman must be *deeply* under the influence of the anaesthetic. In a certain number of cases, however, it is extremely difficult to dilate the cervix. In at least five cases on the table that was so ; in two the woman died undelivered, and in the other three very shortly after delivery. From

¹This also has been my experience in private practice with cases when the convulsions began in the second stage.

these and other cases we are convinced of the great shock that follows forcible dilatation of a very rigid os. Doubtless this shock is much greater when the patient is not deeply under the anaesthetic, but more than once we have seen it occur even when she was. Besides, there is great delay, and valuable time is lost. If the os is extremely rigid, therefore, we think forcible dilatation a dangerous proceeding. Jewett is of the same opinion, and considers it a treatment that affords the woman but little chance of recovery. Now, what is the alternative? We think it is incision of the cervix. By such an operation, in a comparatively short time the uterus can be emptied, and that with very little shock, the blood lost at the operation exercising rather a favourable influence than otherwise. In only one case in this list was that treatment adopted, and the woman recovered.

Looking now at the cases that occur before labour has commenced, the first thing that strikes one is that in a certain number of the cases the disease was arrested. In five of our cases, as we have already stated, that was so. All these cases, except one which had large quantities of fluid by the mouth, were treated by saline transfusion as well as with certain drugs. The most striking case was No. 1, which, as it made a strong impression on all who had anything to do with it, we make no apologies for giving in detail.

Mrs. D., primip., was admitted Aug. 20 of this year, at 2 a.m., deeply comatose. She had had, before admission, eight fits in rapid succession. Prior to these fits, for 20 hours she had complained of severe headache and vomiting, and impairment of vision.

On admission she was found to be deeply comatose, and with a pulse intermittent and very rapid, 140 per minute. She was about eight months pregnant, and was not in labour.

Immediately after admission she had a fit. She was then given an ounce of sulphate of magnesia, $\frac{1}{2}$ gr. morphia hypodermically, a hot pack, and two pints of saline solution into the cellular tissue beneath the mamma. No attempt was made to dilate the cervix.

The pulse immediately after the saline transfusion began to improve, and in six hours the woman began to be restless.

In the evening, although still very drowsy, she opened her eyes when we shouted to her. The following day she was very much better, and passed large quantities of urine. She steadily improved, and 60 hours after admission was delivered of a macerated foetus without even a threatening of convulsions. Now, having seen that case, the prognosis of which all present thought very unfavourable, we are quite convinced that if attempts had been made to dilate the cervix when the woman was admitted, she would have almost certainly died, for her pulse was very bad indeed.

We see that Fitzgerald, in his report the other day of six cases of eclampsia, mentions that in two the course of the disease was arrested. He used diuretics and morphia hypodermically.

We are of opinion, therefore, from these and many other cases we have referred to previously, or read of, that a medicinal treatment, especially the transfusion of saline solutions, should first be tried. If it is going to succeed, in a very short time improvement of the woman's condition will be observable, as witness the case described, so that very little time will have been lost even if more active measures are necessary.

If the expectant treatment fails, or if it is deemed necessary that the uterus should be emptied at once without giving it a trial, there are three courses open: (*a*) forcible dilatation; (*b*) incision of cervix; (*c*) Caesarean section.

The first, forcible dilatation, is the one to be followed if the os is at all soft and dilatable. A very good example of the success of the treatment is the case briefly described in the footnote on p. 58, where the whole operation only took about an hour and a half.

If, however, the os is very rigid, forcible dilatation, as we have already said, has been followed by very bad results, and we would therefore not recommend it. Haultain is a very strong supporter of this operation, but he does not mention the difficulties when the os is rigid. If rapid emptying of the uterus is deemed necessary, when the os is rigid and cannot be dilated, the ideal operation is Caesarean section, one would imagine. Halbertsma was the first to perform Caesarean

section for eclampsia; he reported his cases in 1889, but he first performed the operation in 1878. The results have not been quite as good as the first few cases gave promise of. In 1894 Döderlein was able to gather together the reports of 19 cases, and of these he found 11 mothers recovered and 8 died. Last year Hillman collected 40 cases, with 19 recoveries and 21 deaths; while, as regards the children, there were 23 delivered alive and 18 delivered dead. Since Hillman's figures appeared several cases have been reported. Olshausen the other day stated that he had performed the operation thrice, with two recoveries; Czempin at the same time mentioned a case, and Klein and Martin have also reported cases, the latter a success.

We may say at present that the mortality is about 50 per cent. That of course is a very high figure, but in considering these results it must be remembered that the operation is only performed, and should only be thought of, in very severe and unfavourable cases, when the rigidity of the cervix is extreme and the urgency for evacuation of the uterus most pressing.

Short, however, of Caesarean section there is another operation worthy of consideration, and which we have already mentioned, viz., incision by the cervix (hysterotomy).

This operation for eclampsia is of some age. Velpeau advocated it, and Godema (we quote from Zweifel) recommended it in 1841; indeed in one case this operator completed the delivery in two and a half minutes after having made two deep incisions.

In the last few years the strongest advocate of vaginal hysterotomy has been Dührssen. Few, however, care to follow his recommendation of making incisions as soon after the first fit as possible, for, as Zweifel very rightly remarks, it is impossible to tell after the first fit what course the disease is going to run. Still less would they care to make extensive incisions into vagina and perineum as he so lightly speaks about. Moderate incisions are what recent writers, such as Bidder, Zweifel, and Fehling, recommend.

Zweifel, in supporting the treatment of cervical incisions as against Caesarean section, maintains that by the former

operation the uterus can be emptied just as quickly as by the latter.

If the incisions are to be at all extensive, the operation is carried out most safely by first applying long pressure forceps to the cervix and cutting between them. The great danger is from bleeding, and although Dührssen makes light of it, most others speak of it as often being very real. After delivery the incision must be carefully stitched, and douching or plugging resorted to if the bleeding continues.

The operation must of course be performed with the most thorough antiseptic precautions, indeed, if one might say so, with more than usual precautions, because, according to Olshausen and Zweifel, a very little septic infection and rise of temperature is apt to aggravate the disease.

Prophylactic Treatment.

To complete the consideration of the treatment of eclampsia, we desire to say just a word respecting prophylaxis. We could give many examples of very extreme albuminuria and oedema being relieved, and even eclampsia being apparently warded off by suitable treatment. Regarding the latter point, however, it must be remembered that only a very small proportion of cases of albuminuria and anasarca are seized with convulsions—Fehling, whose figures are the latest, says only 5 per cent.

The treatment of albuminuria in pregnancy is absolute rest in bed, large quantities of milk, and milk and soda or skim milk, and two regular watery evacuations from the bowels with Epsom or other salts. If such treatment does not improve the conditions, or if at any time matters become worse, the question of induction of premature labour should be at once considered.

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No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
1	J. H.	(?)	17	Puerperium	Full time	3	—	Headache and œdema for three weeks
2	A. D.	1	21	Pregnancy (probably)	7 months	23	Albumen, $\frac{7}{8}$ column	Edema of face and ankles, bleeding from nose for three weeks
3	A. R.	1	21	—	6 months	16	Much albumen	Edema of feet and legs for a week before
4	Mrs. C.	—	26	Pregnancy	Full time	2	3rd day albumen $\frac{1}{4}$, hyaline tube-casts	Headache, œdema for four months
5	E. K.	1	18	Labour 1st stage	Not full time	Several	Large quantity of albumen	Edema of face and legs, headache for two months
6	Mrs M'F	9	37	—	Full time	2	Copious albumen, tube-casts (granular and hyaline)	Edema of legs, arms, and face for 3 weeks, headache, dimness of vision, scanty urination for a week
7	M. D.	1	19	Pregnancy	Full time	5	Albumen, $\frac{2}{3}$ column	Edema of face and legs for months, also headache for three days
8	J. S.	1	18	Labour 1st stage (probably)	Full time	15, all before delivery	Albumen $\frac{1}{4}$ (following day)	—
9	J. P.	2	20	Pregnancy	7½ months	9, all before delivery	Albumen and granular tube-casts	Edema of face and legs, ascites for several weeks, dimness of vision and then blindness four hours before delivery

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Chloral, Pot. Bromide	No interference	Recovery	Recovery	Child had several convulsions next day
Pot. Bromide, Chloral, Mustard poultice to neck	Memb. ruptured, Forceps later	Death	Dead	22 before delivery; 1 after. Died 7 hours after delivery
Chloral	No interference (?)	Recovery	Dead	3 fits after delivery
Chloroform, Chloral	Forcible dilatation of os, Forceps	Recovery	Well	No fits after delivery
MgSO ₄ , ChCl ₃ , Pot. Bromide	Podalic Version and Extraction	Recovery	Well (premature)	1 fit before delivery, several shortly after, seizure also on 3rd day of the Puerperium
Pot. Bromide, Chloral	Podalic Version and Extraction	Death	Died on 4th day of convulsions	Died soon after delivery. P.M. Pregnancy kidney, heart fatty
—	ChCl ₃ , Forceps (os fully dilated)	Recovery	Well	4.8.88 one fit; 6.8.88 two fits; 7.8.88 two fits before admission. No fits after delivery. Edema very marked early in pregnancy, but improved under treatment
Cupping in loins, Croton Oil	Dilatation with Barnes' bags, forceps	Recovery	Dead	No fits after delivery
ChCl ₃ , Chloral Veratrum Viride	Insertion of bougies	Recovery	Dead	Fits occurred 26 hours after admission. Patient had been put on milk diet. Child born 36 hours after cessation of fits, labour being induced with bougies

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
10	Mrs M'G	2	33	Pregnancy	7 months	12 in all ; 6 before and 6 after delivery	Albumen $\frac{1}{4}$. No tube-casts	—
11	Mrs. S.	5	25	Pregnancy	6 months	"Several"	Urine solid on boiling	—
12	Mrs. D.	9	35	Pregnancy	7½ months	8, all before delivery	Copious albumen	—
13	Mrs. S.	4	26	Pregnancy	8 months	12	Solid on boiling	Headache for month before, dimness of vision, vomiting, photophobia shortly before fits came on
14	M. S.	1	19	Labour 1st stage	7 months	2	—	Headache
15	Mrs. B.	1	40	Pregnancy	8 months	12	Solid on boiling; tube-casts	Œdema of face and legs for weeks, headache from day before
16	S. G.	1	20	Puerperium	Full time	12	"Little albumen"	—
17	M. M'G.	1	22	Pregnancy	Full time	Several	Copious albumen, casts	Œdema for months, later headache and vomiting for 24 hours
18	Mrs. F.	1	19	Pregnancy	Full time	Very many	Albumen	—
19	Mrs. B.	1	18	—	Full time	—	—	—

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
ChCl ₃ , Chloral, Pot. Bromide	Forcible dilatation, Podalic Version	Recovery	Dead	6 fits before delivery. Very rigid os. 12 hours after delivery fits again started to number of 6
Pilocarpine, Chloral, ChCl ₃ Pot. Bromide	Forcible dilatation, Perforation, Delivery	Death	Dead	Convulsions in last 3 pregnancies. Pilocarpine employed with bad results, died shortly after delivery. Great difficulty in dilating, os so rigid
ChCl ₃ , MgSO ₄ (No mention otherwise)	Forcible dilatation of os and Forceps	Recovery	Well	ChCl ₃ pushed. Under influence for hours during period of seizures. No fits after delivery
Salts, Jalap, Chloral, Bromide, Hot pack	No interference	Recovery	Well	Seizures in all previous labours. 1 fit before labour, 12 during labour. None after delivery
ChCl ₃	Forcible dilatation, Podalic Version, and extraction	Recovery	Alive (but died 24 hrs. after)	No attacks after delivery
Veratrum Viride, Bromide, Chloral	Forcible dilatation, Barnes' bags, Version	Death	Dead	Veratrum viride pushed. Died 9 hours after delivery. P.M. Pregnancy kidney probable; liver healthy but pale; lungs congested (Hypostatic). Membranes of brain œdematous
KBr, Chloral, ChCl ₃ , Hot pack, Jalap, Liq. ammon. Acetatis	No interference	Recovery	Alive	Fits commenced 5 hours after delivery
Chloral, Bromide, Jalap, &c.	Dilatation fingers and Barnes' bags, Perforation	Death	Dead	Died 24 hours after delivery. P.M. Septic peritonitis; extensive laceration of cervix
Hot packs, Diuretics, Chloral	Forcible dilatation of cervix, Version, Extraction	Very ill when left hospital	Dead before operative treatment commenced	Admitted to Hospital comatose. No fits after delivery, but convulsive movements. Paralysis of left arm and leg. Sent to Royal Infirmary where she developed Phlegmasia
	Forceps in cavity	Recovery	Alive	No details of this case. Journal says, "slight eclampsia"

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
20	Mrs. F.	1	23	(?)	Full time	13	Copious albumen	—
21	Mrs. B.	1	21	Labour 1st stage (probably)	8 months	2	(?)	Headache for week before, then very severe and associated with vomiting just before convulsions
22	I. M'L.	1	24	Labour 1st stage	Full time	3	Copious albumen, blood-casts	Headache, vomiting for some days
23	Mrs M'M	2	20	Labour 1st stage	Full time	6	Copious albumen	—
24	Mrs. F.	2	29	Pregnancy	(?)	5	Copious albumen, blood-casts	Œdema of legs, headache for some time, epigastric pain, nausea and increase of headache few hours before fits
25	A. H.	1	23	Labour 2nd stage (probably)	Full time	4	Copious albumen, casts	Œdema of feet, legs, and face for some time, also headaches for two weeks
26	M. S.	1	—	Labour 2nd stage	Full time	3	Albumen	—
27	Mrs. L.	1	—	Labour 2nd stage	Full time	3	Copious albumen	Œdema only
28	Mrs. K.	1	29	Puerperium	8½ months	3	Trace of albumen before, increased after then rapidly disappeared	Headache, œdema of upper eyelids shortly before fits

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Croton Oil, KBr, Chloral, Morphia (once)	Os dilated, fingers and Barnes' bags, Forceps	Death	Dead	Died 4 hours after delivery. No fits after delivery. No P.M.
KBr, Chloral	Forceps (when os fully dil- ated)	Death	Dead	Died 15 hours after delivery. No P.M. Fits only before delivery, but was very rest- less after delivery
Chloral	Contracted pelvis, Craniotomy	Death	Dead	Died within 12 hours after delivery; fits only before de- livery. P.M. Large white kidney; also large quantity of brownish fluid in abdom- inal cavity
ChCl ₃ , and Chloral, KBr	Forceps (when os fully dil- ated)	Recovery	Alive	No fits after delivery
Croton Oil, ChCl ₃ , Chloral, Hot pack	Attempted to dilate os	Death	Dead	Very rigid os. Died undelivered
ChCl ₃ Chloral	Forceps (when os fully dil- ated)	Recovery	Alive	3 fits before delivery; 1 fit half hour after
ChCl ₃ Chloral	No interfer- ence	Recovery	—	First fit just before delivery; 2 shortly after. ChCl ₃ for 7 hours
ChCl ₃ only	Forceps (when os fully dil- ated)	Death	Dead	ChCl ₃ inhalations for 6 hours after delivery; 1 convulsion before, and 2 after delivery; died on 2nd day, when had abortive seizures; urine loaded with albumen the whole time
Chloral and ChCl ₃	No interfer- ence	Recovery	Alive	3 seizures occurred on 5th day

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
29	Mrs. S.	1	38	Pregnancy	Premature	1	Copious albumen	Edema
30	Mrs. S.	1	19	Labour 1st stage (probably)	Not full time	11	—	—
31	A. C.	1	18	Labour 1st stage	Full time	3	Albumen	—
32	M. D.	1	19	Labour 1st stage	Full time	4	Almost solid on boiling	None
33	Mrs. H.	4	28	Labour 1st stage	Full time	2	Trace of albumen	None
34	M. B.	1	19	Labour 1st stage	Full time	41 (only 4 before delivery)	—	—
35	A. M.	1	19	Labour 1st stage	Full time	14 or more	Scanty urine solid on boiling	Severe headache and vomiting day before admission
36	Mrs. B.	2	28	Puerperium	Full time	6	Albumen $\frac{1}{2}$, casts	Edema, dyspnoea, cough
37	Mrs. M.	1	22	—	Full time	8-12	No urine to examine	Headache, loss of sight day before
38	A. C.	1	19	Pregnancy	Full time	About 8	Solid on boiling	Edema two months. Severe pain in epigastrium and head. Dimness of vision for two hours prior to first fit.
39	M. H.	1	19	Labour, 1st stage	Full time	6 in all; 5 in labour 1 after delivery	Fair quantity	Albuminuria of some time. Five hours before first fit severe epigastric pain

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Croton Oil, Hot pack, Jalap, Pilocarpine	Bougies inserted, Barnes' bags, Craniotomy	Death	Dead	Almost entire suppression of urine. Comatose throughout. Died on 2nd day
ChCl ₃ and Chloral	Os dilated fingers, and Barnes' bag, Forceps	Death	Twins (premature) both dead	Very rigid os. No fits after delivery but great restlessness; died suddenly on 3rd day. No P.M.
ChCl ₃ and Chloral, Hot pack	Os dilated, Craniotomy	Recovery	Dead	No fits after delivery
ChCl ₃ and Chloral	Forceps	Recovery	Alive	1 fit before, 3 slight fits shortly after delivery. Pyrexia, restlessness for next four days
—	Os dilated and Forceps applied	Recovery	Alive	No fits after delivery. P.P.H.
ChCl ₃ , Chloral, Pilocarpine	Os dilated and Forceps applied	Death	Alive	Majority of fits after delivery. Died 48 hours after delivery. No P.M.
Chloral, KBr	Os dilated, Version and Extraction	Death	Alive	Comatose throughout. Died shortly after delivery. P.M. Kidneys, pregnancy kidney probably. Hyperæmia of brain. Liver showed fatty infiltration. Stomach greatly distended with gas
Veratrum Viride, Chloral and Bromide	Forceps at outlet	Recovery	Alive	Fits commenced shortly after delivery. Veratrum viride (4 injections) without benefit. Chloral and Bromide at once quieted the pulse
Chloroform	Os dilated gradually	Death	Dead	Os very rigid; great difficulty in dilating. Became greatly asphyxiated during dilatation on two occasions. Died on second attack
Chloral and Bromide per rectum. Chloroform inhalations	Os dilated easily, delivery by Forceps	Death 12 hours after delivery	Dead	After delivery one fit, then gradually sank and died
Chloral and Bromide	Delivery with Forceps when os had become fully dilated	Recovery	Alive	—

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
40	E. M'L.	1	16	Labour, 1st stage	Full time	10 in all ; 8 in labour, 2 18 hours after delivery	Loaded with albumen	None
41	Mrs. B.	1	28	Pregnancy	8 months	6 during pregnancy	Large quantity	For 10 days cedema and for 3 sick and vomited, giddy amaurosis but little headache
42	Mrs. F.	1	23	Pregnancy	8 months	4 fits, all before delivery	Almost solid on boiling. No casts	Severe headache and epigastric pain ; also cedema for several days
43	Mrs. S.	1	28	Just about time when labour began	Before full time	13 in all ; 10 during labour, 3 after, and many abortive seizures	$\frac{1}{8}$ column albumen. Tube casts, epithelial and granular	Headache and cedema for some months
44	Mrs. B.	5	29	About the commencement of labour	Premature	Several during labour, none after delivery	Albumen almost solid on boiling. Granular tube-casts	Severe frontal headache for months before
45	Mrs. G.	—	27	Pregnancy just before labour commenced	6 months	12 fits in all	Albumen almost solid, blood	Felt out of sorts for some days
46	Mrs. L.	2	—	Probably just before labour commenced	—	6 fits, all before delivery	Albumen large quantity	—

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Chloral and Bromide	Os dilated from size of 2/ and delivered with Forceps	Recovery	Alive	Fits after delivery; did not occur until 18 hours after
Large quantities of Fluid, Pilocarpine	Labour completed with Forceps when os fully dilated	Recovery	Alive	On 21.11.95 sick and vomited. On 24th had convulsions and recovered, on 27th had 5 convulsions, then brought to Hospital. One month later delivered, no fits. Whenever quantity of urine passed diminished, woman not so well
Pack. Fluids, Tinct. Veratrum Viride 5 min. every ½ hour, in all 25 min. before delivery	When os size of 1/ dilated with fingers, Version and Extraction	Recovery	Alive	All fits before labour; four occurred in about 12 hours. Child died on the tenth day after having had a series of convulsions spread over several days. The first was on the third day.
Before delivery Chloroform inhalations. After Tinct. Ver. Viride and then Chloral and Bromide	Forceps when os fully dilated	Death	Dead	Tinct. Ver. Viride lowered the pulse rate and seemed to check seizures for a time, 30 minims were given hypodermically. Pulse became more rapid again and coma more profound. Died about 36 hours after delivery
Chloral and Bromide	Os dilated with fingers, Version performed and child extracted	Recovery	Dead	No fits after delivery, but on third day patient extremely restless, so Chloral and Bromide administered
Chloral and Bromide	Attempts were made to dilate os but this failed Woman died undelivered	Death (undelivered)	Dead	Os in this case was extremely rigid. De Ribes bag burst in process of dilating, and even after small excision cervix could not be dilated. P.M. Liver extremely hyperæmic and contains numerous hæmorrhages, mitral valves narrowed; kidney very hyperæmic
Tinct. Ver. Viride, Chloral. Then Bromides as still restless	Os forcibly dilated, Version, extraction, but head stuck in cervix	Recovery	Dead	No fits after delivery

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
47	E. M'K.	1	17	Puerperium	Full time	2	Albumen considerable	—
48	Mrs. K.	1	28	Pregnancy	Premature	8, all before delivery	Loaded with albumen	—
49	Mrs. D.	1	24	Probably just before labour commenced	Full time	6, all before delivery	Loaded with albumen	—
50	L. R.	1	18	—	Full time	9 in all, 8 before and 1 after delivery	—	No premonitory symptoms
51	Mrs. B.	1	28	Pregnancy (probably)	Premature	Several before delivery, none after	Solid on boiling	—
52	Mrs. D.	1	24	Pregnancy	7 months	3	Almost solid, hyaline tube casts	Headache for month, greatly increased in severity, associated with vomiting few hours before first fit
53	K. T.	1	23	Labour 2nd stage	Full time	3	Considerable albumen	Edema for six weeks
54	Mrs. Q.	1	23	Pregnancy	Premature	17 (ten fits before)	Highly albuminous	Edema for two months, restlessness for two weeks headache and blindness for a few hours

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Bromides. Tinct. Ver. Viride. Sal- ine Injection, Pack.	Labour had been com- pleted with Forceps	Recovery	Alive	The first fit occurred immediate- ly after delivery, the second the day following. Both were preceded by great restlessness. Saline transfusion used for first time
Tinct. Ver. Viride, Chlo- ral and Bro- mide	When os had dilated somewhat, dilatation continued; Version, ex- traction	Death	Dead	50 min. Tinct. Ver. given hypo- dermically, also 45 grs. Chloral per rectum. Before death pulse became very rapid and cyanosis became marked
Venisection. 10 oz. blood withdrawn Saline Trans- fusion, Pack	Os forcibly dilated with fingers; Version, ex- traction	Death	Dead	Became very restless after de- livery so 2 drachms Bromide and later $\frac{1}{2}$ gr. Morphia hypo- dermically. Slept for 3 hours then became weaker and gradually sank
Tinct. Ver. Viride	Os dilated, child extracted after Version	Death	Dead	35 min. Tinct. Ver. Viride given hypodermically in 18 hours, reduced pulse rate, but not the restlessness after delivery. One drachm Bromide did this. Pulse became gradually more feeble and rapid
Tinct. Ver. Viride. Sal- ine Trans- fusion	Forced dila- tion of cer- vix. Forceps	Recovery	Dead	Was very much collapsed after delivery
Tinct. Ver. Viride. Sal- ine Trans- fused. Fluids, Chloral	Os dilated. Forceps	Recovery	Dead	Admitted Hospital 14.5.98, two fits before one after admission. Greatly improved under treat- ment, and seven days after delivered without fits
Morphia and mixture of Digitalis, Acetate of Potash, etc. Two pints Saline per rectum	Second child extracted with Forceps	Recovery	Twins (alive)	Severe P. P. H. and became very collapsed. No fits after delivery
Tinct. Ver. Viride. Mor- phia	Forcible dila- tation with bougies. Os very rigid	Death	Dead	This patient had ten fits be- fore delivery, and the <i>fits after delivery did not occur until the fourth and fifth day of puer- perium.</i> On latter day patient died

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
55	Mrs. M'W.	1	21	Before labour (probably)	6 months	7, all before delivery	Large quantity	Edema for weeks. Severe occipital headache some hours before first fit
56	Mrs. D.	1	27	At the beginning of labour (probably)	—	6, 5 before 1 after	Solid on boiling. Epith. and granular tube casts	Edema for a month, also occasional disturbances of vision
57	Mrs. K.	4	30	Before labour (probably)	8 months	3	Abundant albumen, and fatty casts	Headache and swelling of feet for fully a week
58	A. W.	1	19	Before labour (probably)	7 months	14	Abundant albumen	Continual vomiting during pregnancy
59	Mrs. B.	1	27	Pregnancy	Premature	13, all before delivery	Albumen solid, also blood	For two weeks edema and great sickness and vomiting
60	Mrs. G.	2	26	At beginning of labour (probably)	Full time	Several	Loaded with albumen, also blood.	None
61	Mrs. W.	1	26	Pregnancy	—	2	Solid on boiling	Headache, sickness and vomiting for three weeks. Edema for ten days
62	Mrs. M'G.	1	23	Pregnancy	Premature	Several	Almost solid on boiling, with Epith. casts	Edema for two weeks

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Morphia 1½ grs. hypodermically in six hours	Os forcibly dilated	Death	Dead	Was comatose when admitted and remained so. Pulse never below 140
Morphia, Saline Transfusion, Pack	Forceps. Os dilated from half full dilatation	Recovery	Dead	—
Morphia, Saline Transfused. Venisection. Fluids, Hot Pack	Delivered with Forceps without any dilatation	Recovery	Dead	No fits after delivery
Transfusion, 54 oz. in all, Pack. (No effect)	Os from being slightly dilated was fully dilated with fingers. Forceps	Recovery	Dead	Although there were 14 fits before admission there were none after
Transfusion, Venisection, Morphia	When os began to dilate, forcible dilatation then craniotomy and extraction	Death	Dead	Died day following delivery. P.M. Lungs œdematous and patches of consolidation. Kidneys hyperæmic (pregnancy)
Morphia Transfusion	Forcible dilatation with os partly dilated. Version and extraction	Death	Dead	Patient very much collapsed after delivery. Died soon after. Extensive tear made in cervix
Saline Transfusion. Milk and Imperial drink	Not delivered	Recovery	—	Day following admission greatly improved. Albumen by fourth day almost gone. She left then with still some headache and vomiting
Saline Transfusion, Chloral Hydrate	Forcible dilatation. Version and small incisions in cervix. Perforation of head	Recovery	Dead	No fits after delivery but several attacks of twitchings of arms and face

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
63	Mrs. T.	1	23	Pregnancy	8 months	9	Albumen $\frac{3}{4}$	—
64	Mrs. M.	2	38	Pregnancy	6 months	9	—	Oedema of feet for two months
65	Mrs. M'L.	7	29	—	Full time	"numerous"	Albumen almost solid	—
66	Mrs. B.	2	20	Pregnancy	6 months	28 in all; 10 before 18 after delivery	Solid on boiling	Violent frontal headache, two days' nausea and vomiting
67	J. M'K.	1	17	Pregnancy	Full time	14 in all; 10 before 4 after delivery	Small quantity	Severe occipital headache
68	C. S.	1	16	Labour 2nd stage	Full time	7 in all; 2 before 5 after delivery	Albumen	—
69	M. C.	1	22	Before labour (probably)	Premature	15 in all; 10 before 5 after delivery	Large quantity	—
70	Mrs. L.	2	20	Puerperium	—	15	Haze	—
71	E. D.	1	18	Just before labour (probably)	Mature	9	—	—
72	Mrs. D.	1	22	Labour 1st stage	—	5	—	—

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Saline Transfusion, Chlor. Hydrate	Cervix dilated with fingers after dilatation had started. Forceps.	Death	Dead (macerated)	This patient was progressing very favourably when she suddenly died on the 6th day of puerperium. P.M. showed a ruptured duodenal ulcer
Saline Transfusion, Tinct. Ver. Viride	Forceible dilatation. Version, perforation of after-coming head	Recovery	Dead	This patient made a good recovery even although acute pneumonia occurred on 4th day of puerperium. There were no fits after admission to hospital
Saline Transfusion, Tinct. Ver. Viride	Os dilated from size of 2/6. Version, extraction	Recovery	Dead	This patient was unconscious on admission. Urine of child day after delivery was highly albuminous
Tinct. Veratrum Viride little effect, but Chloral had. Saline Transfusion	Labour induced accouchment force, Version	Recovery	Dead	This is a most striking case of recovery. Patient at worst had pulse of 150
Saline Transfusion, Tinct. Veratrum Viride	Os dilated, child extracted by Forceps	Recovery	Dead	10 fits before delivery in 8 hours, and 4 fits after delivery in 6 hours
Saline Transfusion, Tinct. Ver. Viride	Forceps	Recovery	Alive	Pelvis slightly deformed. C.D. 4½ in.
Saline Transfusion, Tinct. Ver. Viride	Os forcibly dilated from size of 1/. Craniotomy	Death	Dead	Ten fits before delivery five after, the first coming on 24 hours after delivery. P.M. Liver markedly degenerated
Saline Transfusion, Magnes, Sulph. fluids, Morph., Chloral Brom., Verat. Viride		Recovery	—	Treatment here seems to have been very varied. The woman very much collapsed but recovered. 1st fit 4 hours after delivery
Saline Transfusion, steam baths	Forceps. Adherent membranes retained	Death	Dead	Shortly after delivery of placenta, pulse began to flag and patient expired
Saline Transfusion, Chloral and Bromide by stomach tube	Os forcibly dilated. Forceps	Recovery	Alive	No fits after delivery. Contracted pelvis, C.V. 3½ in.

No. of Case	Name.	No. of Birth.	Age.	Onset of Fits.	Period of Pregnancy.	Number of Fits.	Condition of Urine.	Prodromal Symptoms.
73	Mrs. M ^H .	1	31	Labour, 1st stage	About full time	19 in all ; 8 before 11 after delivery	Solid on boiling, blood	None
74	Mrs. A.	4	30	Pregnancy	8 months	4 in pregnancy	Very albuminous	None
75	Mrs. L.	1	21	Pregnancy	Full time	20 fits before admission in a period of 18 hours	Albumen $\frac{1}{2}$	Cedema for two weeks
76	Mrs. D.	1	25	Pregnancy	7 months	2	Albumen abundant	Headache, vomiting, cedema
77	Mrs. L.	6	30	Labour, 1st stage	6 months	7	Albumen $\frac{3}{4}$ column, blood, tube casts	Headache, vomiting for day before
78	Mrs. D.	1	30	Pregnancy	8 months	9	Albumen	Dimness of vision, headache, vomiting for 20 hours
79	Mrs. C.	4	28	Labour, 2nd stage	—	11 in all ; 3 before 8 after delivery	Albumen $\frac{1}{2}$ column, blood and casts	Headache and vomiting for short time before
80	Mrs. F.	1	—	Labour, 1st stage	Full time	7 before and 2 after delivery	Albumen abundant	—

TREATMENT.		Result to Mother.	Result to Child.	REMARKS.
Medical.	Operative.			
Saline Transfusion	Forcible dilatation from $1\frac{1}{2}$ in. diameter. Both extracted with Forceps	Death	1st dead 2nd alive	Twins. This patient was treated by salines entirely. During three days she got 406 oz. but only passed 280 oz. At post mortem large quantity of fluid in tissues and abdominal cavity
Saline Transfusion and fluids	—	Recovery. Labour 3 days after	Dead	Labour came on 3 days after last fit. There was no trouble with it
Saline Transfusion, Chloral and Bromide	—	Recovery	Dead	Labour did not commence until 18 hours after admission. No fits after admission
Saline Transfusion, Magnes. Sulph.	Os forcibly dilated. Version	Recovery	Dead	Had been treated for three weeks prior to admission for acute nephritis. The urine on dismissal 10th day contained albumen
Saline Transfusion, Morphia hypodermically	Forcible dilatation. Version	Death	Dead	Os extremely rigid and great difficulty in dilating cervix. P.M. both kidneys full of small cysts
Saline Transfusion, Morphia hypoder. Pack	—	Recovery	Dead (macerated)	Labour came on some 48 hours after admission. On admission she was comatose, and the pulse was intermittent. 140
Saline Transfusion, Morphia. Pack	—	Recovery	Alive	Delivered outside
Saline Transfusion. Steam bath salts	Forceps	Recovery	Alive	First four fits within four hours. First post partum fit four hours after delivery.

CHRONIC INVERSION OF THE UTERUS, WITH NOTES ON THREE CASES.

By JOHN EDGAR, M.A., B.Sc., M.B., C.M., F.F.P.S.G.,

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SINCE November, 1899, three cases of chronic inversion of the uterus have come under my notice, two of them due to a sessile fibro-myoma attached to the fundus uteri, the third a puerperal case. As this complication is now rare, and the cases present some features of interest, I have considered it right to place them upon record.

CASE I.—*Complete procidentia of completely inverted uterus due to a fibro-myoma attached to the fundus uteri; Küstner's operation; vaginal hysterectomy; recovery.*

Mrs. M'L., æt. 35, 3-para, was admitted into the Samaritan Hospital on 9th Nov., 1899, with the complaint that "the womb had been coming down for fifteen months."

History.—All three labours normal; last one $4\frac{1}{2}$ years previously. Menstruation always profuse. For past fifteen months had felt a small lump at vulvar orifice, disappearing on lying down, and with no projecting tumour on it such as was found at fundus on admission. On 14th Sept., 1899, had a severe flooding, which ceased on 15th. On 19th the large mass present on admission came down suddenly. She had not been straining, nor, except for a bearing-down feeling, was there at any time any pain. Profuse watery discharge during the whole fifteen months. Micturition difficult since 19th Sept.

Condition on admission.—Anæmic. So morbidly nervous that she refused to be examined unless chloroform was given,

though she stated that the mass was not at all sensitive. This mass was then seen to be the completely inverted uterus *plus* vagina, with a sub-mucous fibro-myoma at the fundus. (See photograph taken by Dr. Frew.) Measurements: Total length, 7 inches; length of uterus, $4\frac{3}{4}$ inches; length of corpus uteri, $3\frac{1}{8}$ inches; circumference at fundus, $6\frac{1}{4}$ inches, at isthmus, $5\frac{1}{2}$ inches; width between cornual openings, $2\frac{3}{8}$ inches. The tumour ($1\frac{3}{4}$ by $1\frac{1}{4}$ by $\frac{7}{8}$ inch) was sessile, and attached to the right half of the fundus in front of the right cornual opening by a circular base, one inch in diameter. The diagnosis of fibro-myoma was afterwards confirmed by microscopic examination. The inverted uterus was thickened and dark red, with here and there small pale patches as if the epithelium at these places had become squamous. Consistence firm. The vaginal wall near the cervix was eroded, elsewhere very white, due to proliferation of epithelium.

Treatment.—*11th Nov.* Tumour removed and wound stitched with catgut.

13th and 14th Nov. Ineffectual attempts at reposition of uterus under chloroform; hot douching and tamponade with gauze.

15th Nov. After another fruitless attempt, a Champetier de Ribes' bag was inserted and left in.

16th Nov. Taxis having again failed, a transverse incision was made into Douglas' pouch through the posterior fornix, and the os was dilated with two fingers. No adhesions were found. Another attempt at re-inversion of the uterus having failed, Küstner's operation was completed by making a mesial incision through the whole thickness of the posterior wall of the uterus from 2 cm. below the external os to 2 cm. above the fundus. The uterine walls proving to be too thick and firm to allow even then of re-inversion of the organ, it was finally decided to perform hysterectomy. This was done as follows: A silk ligature was passed through the cervix from behind forwards on each side and tied. Three other silk ligatures were then passed, also from behind forwards, through the intervening portion of the cervix, and, after the uterus had been removed by the scissors, were tied over the stump. There was practically no bleeding.

After removal of the uterus, the anterior and posterior uterine walls were found to be each one inch in thickness and so firm that re-inversion was possible only with great difficulty, and was attended by tearing of the tissue. The fundus was five-eighths of an inch in thickness.

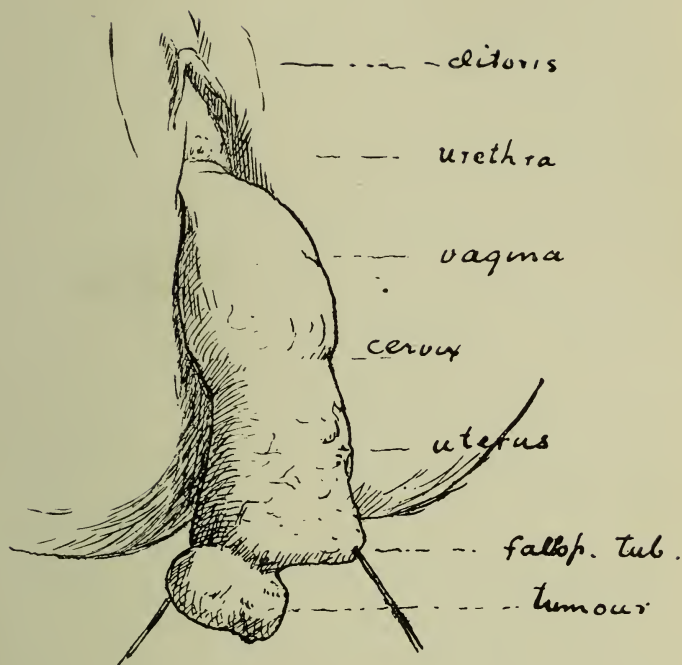
The patient made a good recovery, and has since remained well.

CASE II.—*Case of complete puerperal inversion of the uterus; re-inversion.*

Mrs. K., æt. 29, 3-para, was admitted into the Samaritan Hospital on 27th March, 1900, with a note from a medical man that she had been suffering from "prolapse of the uterus since the birth of a child twelve days before." The duration of the labour was 24 hours, presentation normal, and she was very exhausted, chiefly owing to previous mal-nutrition. Considerable hæmorrhage during the third stage. The midwife in attendance expressed the placenta with considerable force, but patient did not know whether she pulled on the cord or not. Subsequent bleeding normal in amount, and patient felt well except for a bearing-down feeling till the evening of the same day, when, on rising to micturate, the womb suddenly appeared outside with a gush of blood. The midwife, having failed to replace it, called in a medical man, who pushed it back with some difficulty. There was no further hæmorrhage. A week afterwards the uterus prolapsed again. The doctor once more replaced it, and a few days afterwards sent her into the Samaritan Hospital with the note above mentioned. There was no pain throughout.

Condition on admission.—Very anæmic. Frequent micturition. Feeling of bearing-down. Night-sweats and cough; examination of chest showed signs of phthisis pulmonalis. Trace of albumen in urine. On examination the completely inverted uterus was found in the vagina.

Treatment.—On several occasions taxis was employed under chloroform, but with no effect. In the intervals hot douching and firm tamponade with iodoform gauze. A colpeurynter was also inserted into the vagina and filled with mercury, and the patient was kept for several hours lying prone with the pelvis raised. On the last occasion when taxis was tried so



CASE I.

much force was used that the cervix was torn from its connection with the bladder. The rent was stitched and healed by first intention.

On 2nd May, having resolved to make one more effort at taxis, and, in the event of failure, to slit up the posterior wall of the uterus from and including the external os towards the fundus as far as might be necessary, I had the patient anæsthetised, but finding that menstruation had begun and was profuse, I tamponaded the vagina and sent the patient back to bed. At midnight there was a severe flooding, which continued less profusely for several days. Ergot and whisky were administered, and the vagina was douched (120° F.) and firmly tamponaded with iodoform gauze twice daily. On 7th May the uterus was found to have become re-inverted as a result of this treatment. It has remained in normal position since, and the patient has improved in health.

CASE III.—*Complete inversion of uterus due to fibro-myoma on fundus uteri, Hirst's operation; hysterectomy; death from secondary cause 5½ weeks after operation.*

Mrs. M., æt. 48, widow, one child 17 years of age. I was asked by Dr. Gray to see this patient because of metrorrhagia and great debility of 18 months' duration. There were also a bearing down sensation and a profuse watery discharge, but never any pain nor any urinary trouble.

On examination I found the vagina occupied by a mass, which proved to be the completely inverted uterus with a sessile fibro-myoma of the size of an apple situated at the fundus.

Treatment.—On 9th March I removed the tumour, and subsequently repeatedly endeavoured to replace the uterus by taxis, hot douching, and tamponades of gauze, but without effect. As the uterus was still increased in size, I got the nurse to insert glycerine tampons into the vagina after each morning and evening douche, and on 3rd April sent patient home to continue the treatment.

By this time the bearing-down feeling had disappeared. Shortly after her return home, her nurse was compelled to leave her on account of illness. Mrs. M. then got out of bed and resumed her household duties. At the end of her second menstrual period a flooding supervened, and continued with

several fainting attacks until on the fourth day Dr. Gray was called in and arrested it by plugging the vagina and injecting ergotinin hypodermically. On 15th and again on 25th June I made two more unsuccessful attempts under chloroform at reposition of the uterus.

Three days later Mrs. M. was re-admitted into the St. Elizabeth Home. Though she was still weak and very anæmic, we feared another attack of hæmorrhage, and therefore on 5th July Dr. Gray administered an anæsthetic, and, after another ineffectual attempt to re-invert the uterus, I opened into the pouch of Douglas, and, having dilated the os with two fingers (no adhesions found), I once more employed taxis. Failing in this, I slit up the posterior uterine wall, beginning at the external os (Hirst's operation), but found this also ineffectual. I then performed hysterectomy in the manner described in Case I. The reason for the failures in taxis was then discovered to be that several small fibroids were present in the uterine walls, rendering them bulky and stiff.

The patient made a good recovery from the operation. On 28th July, twenty-three days after the operation, I left for Paris. Dr. Gray attended her after this, and reports as follows: "A few days after Dr. Edgar's departure, Mrs. M. suddenly felt a sharp, cutting pain in the right lower axillary region, and her temperature rose to 101° F. Slight dulness and deficient R.M. at this place. Hard cough with scanty mucous spit. Three days afterwards the pain disappeared, but there was still dyspnœa. Cough less frequent, but breathing still rapid and shallow.

"On 8th August she left for home, somewhat improved. Next day the dyspnœa returned; respirations 50, pulse 100, and temperature 102° F., hacking cough, no spit; dulness in right lower axillary region, the site of former pain; R.M. diminished; no crepitant râle nor bronchial breathing; V.S. murmur at apex of heart; no abdominal tenderness nor distension. The dyspnœa and weakness increased, and on evening of 11th, Dr. Edgar, who had just returned home, was called in consultation."

On my arrival I found patient moribund. She was cyanosed and nearly comatose, and died on the morning of the 12th.

A post-mortem examination could not be obtained. The cause of death, therefore, cannot be known exactly, but it was possibly pulmonary embolism. Dr. Gray states that patient, when young, had pulmonary phthisis, and is of opinion that possibly "during convalescence, while patient was still weak and anæmic, a latent tubercular condition developed into an active process, and brought about the fatal issue."

REMARKS,

Inversion of the uterus is a rare affection, and is said to happen most commonly as a complication of the third stage of labour. In the Rotunda Hospital it occurred once in 190,800, and in the lying-in department of the Vienna Hospital once in 250,000 labours.

Etiology.—In puerperal cases it is generally agreed that, provided the uterine wall be relaxed, the determining cause is either traction on the funis, or pressure on the fundus uteri by the hand, or by straining efforts on the part of the patient. Precipitate labour is frequently mentioned as a cause, but that it can be so directly I deny, inasmuch as the active contractions would prevent rather than cause inversion of the fundus. Indirectly, however, as a cause of atony of the uterus in the third stage, it may predispose to this complication of labour.

In non-puerperal cases, opinions as to the etiology are not so unanimous. These cases are always due to the presence of an intra-uterine tumour, which is, as a rule, sessile and situated at the fundus, as in two of the cases above reported. It is generally thought that the tumour, being a foreign body, excites the uterus to contract, and that the contractions result in expelling the growth through the os. If pedunculated or loosely attached, the tumour will not, as a rule, drag the fundus along with it, but if it be sessile and firmly fixed, incomplete inversion will occur, and the lower part of the uterus will then contract on the inverted fundus, drive it down still further, and so bring about complete inversion of the organ. Schauta opposes this view strongly. He says that the contractions of

the uterus would prevent and not occasion an inversion, and considers that the cause is the same as in puerperal cases, namely, (1) relaxation of the uterus due to atrophy and degeneration of the uterine wall, (2) the presence of a cavity in the uterus, such as might be caused by placenta, blood-clots, or tumour, and (3) either (*a*) traction from below by a volsella or even by the weight of an intra-uterine growth, or (*b*) pressure from above by straining efforts, etc. While removing a submucous myoma, whether it be sessile or pedunculated, traction on it may certainly invert a relaxed uterus, and this is a complication which must be kept in mind; but it is not so easy to understand how straining efforts, with or without traction by the weight of a tumour, would be sufficient to force a relaxed fundus through the os, unless the cervix be dilated, as in the third stage of labour. Is it not possible that uterine contractions, excited by the presence of an intra-uterine growth, would have a tendency, by the same mechanism as in labour, to dilate the cervix, and that, once this has happened, the tumour may be so far driven into the os that, when relaxation occurs, the growth may remain caught and so prevent the fundus, to which it is attached, from returning to its normal position? The uterus would then be cupped, and intra-abdominal pressure might subsequently be sufficient to complete the inversion.

Symptoms.—The usual symptoms are (1) hæmorrhage and leucorrhœa, with consequent anæmia and debility, (2) bearing-down sensations, and (3) sometimes urinary troubles. In acute cases there are generally pain and shock in addition to hæmorrhage. Case I. is remarkable in its absence of hæmorrhagic discharge. Though not stated in the report, menstruation, during the three months immediately prior to admission into hospital, had occurred only once, viz., six weeks before, and had then, though profuse, lasted only two days.

Diagnosis.—The typical signs are (1) the presence of a mass in the vagina, or protruding through the vaginal orifice; (2) the absence of the fundus uteri from its usual situation; (3) the presence of a cup-shaped depression above, near which the ovaries can be felt; and (4) the fact that the sound cannot be passed to the normal distance through the os. (5) On careful

examination of the protruding mass, the cornual openings may be found, and the colour is, as a rule, not pale as in the case of a polypus, but red. The presence of a fairly large sessile myoma at the fundus may, however, mask this fifth point in the diagnosis. It is generally stated that the inverted uterus is more tender than a polypus, but I cannot say that I noticed any special tenderness in the three cases above reported.

Though mistakes have been made, they have generally been the result of an imperfect examination. The modern method of removing polypi by torsion and cutting with scissors might conceivably lead to fatal hæmorrhage should the supposed polypus be an inverted uterus. A less dangerous but a very ridiculous error would be the endeavour to push up a polypus under the impression that it was a case of inversion of the uterus.

As showing the difficulty in diagnosis which a case may present, let me quote the following. In January, 1897, a patient was sent to me as a case of inversion of the uterus, due to a sessile fibroid at the fundus. On examination my first impression was to confirm this diagnosis, because (1) the mass in the vagina was of the size and shape that might be expected in such a case, (2) with the finger passed into the cervix along the anterior aspect of the mass, the pedicle and the cervix were found to be continuous at the internal os, and (3) the body of the uterus could not be felt in its usual situation. A sound could not be passed into the uterus behind owing to the bulk of the tumour in the vagina, so that this mode of differential diagnosis, which is recommended in all the text-books, could not be practised. I could not satisfy myself, however, that there was a cup-shaped depression above, so with some difficulty, the patient having been anæsthetised, I passed my finger round the pedicle and into the uterine cavity behind, and managed to reach the fundus, so proving that the mass which occupied the vagina was not the inverted body of the uterus, but a polypus attached by a thick pedicle to the right and anterior surfaces of the corpus uteri. Bimanually I was then able to determine that the uterine body had been dragged backwards and to the left by the tumour. A recto-abdominal examination would have been of assistance had the cervix not been sufficiently dilated to allow the entrance of the finger.

Treatment.—While employing taxis in the three cases reported, I fixed the cervix by volsellæ, and also steadied the uterus by pressure from above. Case II. serves as an illustration of the care which must be taken not to employ too great force. Several cases have been reported in which hot douching and firm tamponade of the vagina with iodoform gauze have been successful. Case II. is a case in point. Possibly the free hæmorrhage aided in some measure by lessening the bulk of the organ, and possibly also by leading to relaxation of the tissues. Though this treatment failed in the other two cases, it is a method which is worthy of a trial when taxis fails in effecting its purpose. Treatment by colpeurynters did not succeed in my cases. In Case II. I tried the method advocated by Halban, Schauta, and others, of inserting a colpeurynter into the vagina and filling it with a heavy plastic material, but did not succeed. Small shot has been recommended for this purpose, but, in my opinion, mercury is the best material. I did not try any of the numerous repositors, such as Aveling's or White's, which have been recommended.

Operative treatment.—In Case I. I tried Küstner's, and in Case III. Hirst's operation. Though unsuccessful, for the reasons mentioned in the reports, I should again try one of these operations, preferably the latter, in any future case I may have under my care, provided it should prove otherwise intractable. Hysterectomy is the *dernier ressort*, unless in cases in which sloughing, gangrene, or cancer is present. In Case I. I should not have resorted to it so soon had the patient been reasonable, but she was so morbidly nervous that she did not permit me to employ milder measures for a sufficient length of time, and the condition was too formidable to allow her to go away *in statu quo ante*. In Case III. the bleeding was so alarming that both Dr. Gray and I considered it unsafe to delay radical measures for the patient's relief. The method of operation which I adopted in these two cases appears to me very simple and satisfactory.

TUBAL GESTATION ; ITS PATHOLOGY AND DIFFERENTIAL DIAGNOSIS, AS ILLUSTRATED BY THE RECORD OF TWENTY-TWO CASES.

By A. LOUISE M'ILROY, M.D.

WITH all our knowledge of tubal gestation, and with all the researches of learned authorities, we have not arrived at any definite conclusions with regard to its *etiology*. The whole question seems to turn upon the point "where does fertilisation take place?" Some writers¹ take it for granted that fertilisation takes place normally in the Fallopian tube, and most probably in its outer half. If this is so, then it is quite easy to understand the theory of obstruction in the tube, and bring forward the whole train of causes, such as tumours, erosions, and inflammations, and conscientiously rest satisfied that we have for ever slain the demon of doubt. On the other hand, we have the theory that "fertilisation normally takes place in the uterus, and that it is accidental when occurring in the tube."²

When the ovum is fertilised it makes for the nearest resting place it can find, and as the uterus seems in every way more suitable for its reception than the tube, it is not unlikely that in the uterus fertilisation takes place. The laws of nature do not admit of accidents, and although they may appear as such, it is only that the veil for us has not been torn aside.

¹ Galabin's *Manual of Midwifery*, p. 47.

² Bland Sutton, *Surgical Diseases of Ovaries and Fallopian Tubes*, p. 242.

In many cases fertilisation does take place in the tube, and this may be due to something preventing the ovum from getting down to the uterus, or to undue activity of the spermatozoon.

But what is this obstruction?

Webster¹ describes a source of obstruction produced by "the ring-like swelling of the deeper layers of the mucosa, which resembles that of the uterine decidua." When the ovum is fertilised, the surrounding structures undergo a change, getting ready, as it were, to adapt themselves for the support and sustenance of the newly formed organism. This obstruction occurs on the uterine side of the tube when the oosperm has settled in the intermediate or outer part.

This view is refuted by Taylor,² who states that no one has been able to detect in the pregnant tube anything approaching to the decidual membrane of the uterus. In my own specimens, which can hardly be parallel, as mine were after rupture had taken place, I found decidual tissue in several on the uterine side of the attachment of the ovum. During menstruation the turgescence of the uterine mucosa may extend slightly along the tube, and thus may cause arrest of an ovum which has just entered the tube before the onset of the menstrual period.

Salpingitis as a cause, although dismissed by many modern writers, has many points in its favour; the thickening of the tube wall, the peritonitic adhesions occluding its lumen, may interfere with the peristaltic action, and so arrest the ovum. But, on the other hand, if it has been far advanced, it will have occluded the abdominal ostium, and so prevent the entrance of the ovum; it may also occlude the uterine end of the tube. In all my cases, I made careful microscopical examination of the uterine end of the tube, and in every one found it patent.

The old theory that a desquamative salpingitis is necessary as a preparation for the settling down of the fertilised ovum is now given up, as most people now admit that it has

¹ *Ectopic Pregnancy*, 1895, pp. 10, 11.

² *Extra-Uterine Pregnancy*, p. 12.

no parallel in menstruation, which is not a preparation for, but a consequence of the failure of pregnancy having taken place.

Salpingitis, not as a cause, but as a factor in its later progress, is more important with reference to tubal gestation. By setting up thickening or atrophy of the tube, it may influence subsequent rupture or tubal abortion.

It is difficult to decide the previous existence of salpingitis, as the pathological condition of the opposite tube is in many cases due to the pregnancy in the other tube; the history is a guide, of course. The age of the patient is the same as in ordinary pregnancy. The following table bears out this as taken from my own cases:

Age.	Age
20-25=3.	30-35=6.
25-30=7.	30-40=7.

Previous sterility is not a factor to be considered in tubal gestation, as shown by my own experience—out of 16 cases:

Last pregnancy 1 year and less than 1 year,	-	-	6
Do. 2	do. 2	do.,	- - 6
Do. 3	do. 3	do.,	- - 2
Do. 4	do. 4	do.,	- - 2

One of these occurred twelve weeks after birth of last child; only two cases were absolutely sterile. Previous uterine abortions may be a factor in the causation by setting up pelvic complications subsequently.

That tubal pregnancy is an abnormal condition is evident from its subsequent course, as no case can go on to full term successfully without operative interference; and it is also evident that the determining factor in its causation is some abnormal arrest of the oosperm or impregnated ovum in its progress towards the uterus from the ovary.

That this arrest may occur in the ovary is theoretically possible, but has as yet to be proved.

That it may occur in the abdominal cavity has been proved impossible on account of the strong absorptive powers of the peritoneal surfaces; cases recorded previously as such being

tubal in origin, and afterwards growing within the abdominal cavity, the embryo being protected by an almost invisible pellicle or amniotic membrane.

Thus we see that all forms of extra-uterine pregnancy are tubal in origin. Some specimens appear doubtful on examination, as the tube has become disorganised, or so incorporated with surrounding structures as to be almost unrecognisable, but on careful examination we will find that the placental attachment is where the tube wall is to be found.

Let us here shortly discuss the early life history of the growing ovum. It is difficult to be accurate with regard to the early stage, as the specimens from which we derive our knowledge have generally undergone some change before operation, either injured by pressure from surrounding parts or from haemorrhage. Classifying them all together, we find a general plan to guide us.

After the ovum has been fertilised it immediately seeks a resting place, and thus becomes engrafted on the tubal mucosa; the surrounding structures become more vascular, the mucosa surrounds and soon covers the oosperm; the chorionic villi develop in the special zone of mucous membrane. During this time the oosperm is increasing rapidly in size, the blood vessels become enlarged, and the tube is stretched and distended, and so compensatory hypertrophy takes place.

If, as referred to before, salpingitis has been present previously, the walls may have become fibrosed, less vascular, and therefore less able to adapt themselves to changes. Thus the elasticity is very slight, and the tube must rupture sooner or later. If, however, the tube wall is able to resist the increased pressure the pregnancy may go on further, and may end as an abortion into the uterine or peritoneal cavities, or as a later rupture of the tube wall.

During this time the uterus has been increasing in size by "sympathetic increase." This is due to increased vascularity, and also to the formation of a membrane in its interior, the "uterine decidua." This is not in direct contact with the oosperm, but is closely related with its life history, as it is shed when anything interferes with the growth of the ovum. Sometimes it is passed entire as a cast of the uterus, but is

generally in shreds mixed with blood-clot. The following case is illustrative :

Mrs. B., aet. 31. History, "one month ago took severe pain in abdomen with inability to defaecate; pain was more severe in left iliac region, accompanied by haemorrhage lasting five days. Menstruation previously regular, but amenorrhoea for five weeks before onset of pain." Patient was pale and collapsed, abdomen distended and tender; dull over pubic region, uterus retroverted, mass in D.P. While in hospital patient took severe "bearing down" pains followed by expulsion of decidua, with copious haemorrhage. She felt great relief after this.



FIG. 1.

The accompanying drawing (Fig. 1) shows the condition. The decidua is triangular, resembling the shape of the interior of the uterus, the base being at the fundus, and the apex at the os internum. The outer surface is shaggy, the inner smooth, showing the glandular openings. The external, or surface next the uterus, is shaggy, and shows large glandular interspaces with decidual cells. This is the spongy layer of the decidua. The cells in these spaces are cubical. In a section, which was taken from the more superficial part, the glandular spaces are smaller, the epithelium being flattened and the interglandular tissue increased in quantity. Large decidual cells were well marked in all the sections.

To return to the tube, we find that the pregnancy will take one of the following courses :

- (1) It will go on growing in the tube, and become either aborted through its ostia or it will cause rupture of the tube by its increased size.
- (2) It will become changed into a *tubal mole*, which will either abort or cause rupture of the tube.

The condition of the ostia of the tube is important, the patency of these predisposing to abortion rather than to rupture. In all my cases of tubal abortion, the abdominal ostium was patent, and in many it was even dilated. I made a careful and minute examination of the uterine end of the tube in all my specimens, and in every one the lumen of the tube was patent, although in several the opening was very small.

Tubal moles only arise in the first two months of pregnancy, and the changes are similar to those which occur in the formation of a uterine mole.

"A *mole* is an early embryo and its membranes, into which blood has been extravasated."¹ They vary in size, the smaller ones being cylindrical and the larger ovoid in shape. The amniotic cavity is usually at one end, due to the pressure of the haemorrhage. The embryo may or may not be present: but it is the exception in my experience to find the embryo in a tubal mole, as it usually has become disorganised by pressure from the surrounding blood-clot, or it has been lost in the peritoneal cavity, when the mole has ruptured at the time of dislocation from the tube. The blood lies between the amnion and the chorion, in what Bland Sutton calls the "subchorionic chamber,"² and he maintains that the blood is derived from the circulation of the embryo, owing to his having found nucleated blood corpuscles in it. But it appears to me incredible that such a small organism as the embryo could be the source of so much haemorrhage. Is it not more reasonable to assume that the inflammatory fibrin covering the outer surface of the chorion, and in whose walls fresh vessels are formed, is a possible source of the blood? The formation takes place by stages, as

¹ Sutton and Giles, 1900, p. 240.

² *Ibid.*, p. 240.

the blood-clot shows a laminated structure. The causation of tubal moles is not quite understood, but the following explanation may deserve consideration:—When the oosperm is in the tube, and more especially when near its abdominal end, it weighs the tube down, dragging it behind the uterus, in which position it is liable to meet with sudden pressure. Then, if its early attachment to the tube wall is slight, there may be some haemorrhage, which is the result of undue exertion on the part of the patient. This exertion is repeated, and thus we have a succession of haemorrhages, until finally there is one attack so severe as to bring the condition under our notice. When the mole is formed it is liable to expulsion into the uterine or abdominal cavities, forming complete tubal abortion. The more usual condition is where the mole remains partially attached to the tube wall, like a polypus.

Tubal moles vary in size, and are generally ruptured before the specimens are obtained. The outer surface or chorion is covered with villi, and the inner or amnion, investing the amniotic cavity, is usually nearer to one pole of the mole. On microscopical examination we will find the presence of villi more easily if we examine either pole, as they are more numerous at the poles. Now we have to consider the probable termination of the tubal pregnancy—the two methods being by:

1. Tubal Abortion.
2. Tubal Rupture.

1. *Tubal Abortion*, or “the partial or complete separation of the ovum from the tube wall, accompanied by haemorrhage into the tube lumen, and the escape of the blood along with part or whole of the ovum into the peritoneal cavity.”¹ This is most likely to occur early in the pregnancy, as after the eighth week the abdominal ostium is usually closed. In all my nine cases the abortion occurred before the eighth week.

“In tubal abortion the oosperm is nearly always converted into a mole,”² but not always, as proved by Case II. It occurs, as stated by most writers, in those cases where the

¹ Webster's *Ectopic Pregnancy*, p. 70.

² Bland Sutton, *Surgical Appliances of Ovaries and Fallopian Tubes*, p. 257.

oosperm is situated near the abdominal ostium. In five of my cases, however, it occurred when the attachment was at the uterine end. It may also occur into the uterine cavity when near the isthmus, but the abdominal form is the most common, as in this case the mole is following the route of least resistance. It also occurs in a healthy tube whose walls are elastic and easily contractile. The mole is discharged with considerable haemorrhage into the peritoneal cavity, giving rise to the symptoms of collapse, etc., and at the same time haemorrhage takes place into the uterine cavity, causing the "discharge or flooding." But in most cases the mole becomes only partially detached from the tube wall, and thus acts as a source of irritation, keeping up a continuous haemorrhage into the abdominal cavity—the characteristic "blood-drip."

Complete expulsion of the ovum is rare, although cases have been recorded by Bland Sutton, Ortheman and others, the difficulty of recognition being in the fact that it becomes lost. The blood passing through the abdominal ostium may be so copious as to cause death, but the more common form is where the blood is gradually poured through the opening, thereby forming a blood cyst or *intra-peritoneal haematocele*. This by a gradual increase of its contents forms an outer or firmer layer of laminated clot, forming the *sac*; it is attached to the fimbriated extremity of the tube, and occasionally by means of its adhesions incorporates the whole tube and ovary in its walls. The lumen of the tube may be shut off from the haematocele cavity, and thus no fresh haemorrhage takes place; the haematocele remains quiescent and becomes absorbed, but more usually the sac in its turn ruptures and grave symptoms supervene. If the abdominal ostium becomes closed, the blood in the tube may suppurate, forming a *pyosalpinx*, or it may increase in size until the tube ruptures; the latter is a very common condition in tubal gestation. The symptoms of tubal abortion are less severe than those of tubal rupture, as the process is more gradual. The patient complains of colicky pains in the abdomen, with irregular uterine haemorrhages, as contrasted with the collapse and shock attendant on rupture, although it must be remembered that

subsequent rupture of the haematocele sac has often very grave consequences, and that it is more often on account of the rupture of this sac that patients come under our notice than on account of the abortion, the history concerning which is often very vague. In describing tubal abortion I refer to the incomplete form. I have endeavoured by the following table to illustrate my remarks by reference to my cases, the pathology of which I have discussed separately at much fuller length.

CASE I.

On Operation, left tube was distended by mole near its abdominal end, and was stretched over top of the haematocele sac, which filled the lower part of the pelvis. The tube ruptured during removal.

Specimen. The tube is in upper part with rupture near its uterine end, where the walls were thinnest; below is mole sectioned to show embryo in its amniotic cavity. The mole was attached near the abdominal end, so that the pressure of the haemorrhage must have caused thinning of the tube walls near the uterine end. It was dark red in colour, cylindrical, and was covered with fresh blood-clot: it measured $2\frac{1}{2}$ in. in length and $1\frac{1}{2}$ in. in diameter. After hardening in formal sol. I found on incision that it contained an embryo of about six weeks, lying in the amniotic cavity, which was about 1 in. in diameter, and surrounded by laminated blood-clot, the latter being in "the sub-chorionic chamber." The ovary was cystic, filled with a jelly-like substance.

Section near uterine end of the tube shows patency of the tube lumen, with erosion of the epithelium in some places.

CASE II.

On Operation, the haematocele sac with the left pregnant tube were found in a mass of blood-clot filling the pelvis, roofed over by omentum.

Specimen. This specimen is of great pathological interest on account of its rarity. The left tube is shown in longitudinal section, containing a mass of placenta and blood-clot within

its unruptured walls; while the cord passing through the abdominal ostium is attached to the foetus which lay in the haematocoele sac. The foetus was in a shrivelled condition owing to the pressure: it had passed through the ostium at an earlier date, the amnion remaining intact, inside which it continued to grow, and protected by it against the surrounding blood-clot—the result of its dislocation from the tube. The pressure from this haematocoele had caused death of the foetus. Haemorrhage had occurred eleven weeks before operation, at which time the abortion may have taken place. The uterine decidua was not expelled until nine weeks later, which may, on the one hand, be a proof that the foetus continued growing after its expulsion from the tube, or, on the other, there may have been a gradual and partial separation of the decidua keeping up the uterine haemorrhage, along with that from the tube. Cases such as this are extremely rare, one has been recorded by Webster (page 56 of his book)—it occurred in the practice of Halliday Croom—and one was reported by Mr. Lawson Tait to the Obstetrical Society in 1892. But in neither of these is the exact process explained by which the embryo was extruded. Webster says, “in a manner unknown, escape of the foetus took place into the peritoneal cavity,” but he suggests a gradual thinning or hernia-like protrusion of the tube-wall, or an escape by the abdominal end of the tube. In my specimen the wall of the tube was absolutely intact, so that the escape must have been through its abdominal ostium. I think this case clears up *this* form of tubal abortion, our knowledge of which was rather vague.

Section of the tube near its uterine end shows erosion, and also flattening of the columnar epithelium, with atrophy of the plicae and increase in the connective tissue of the tube walls.

CASE III.

On Operation, the unruptured left tube was found stretched and distended over the haematocoele sac.

Specimen. The left tube is seen, a slit having been made in its posterior wall in order to show the mole in its interior

attached near the uterine end. Part of the haematocele sac is left attached to the fimbriated end to show its interior, shaggy from the deposit of fibrin. The fimbriae are thickened, and a peritoneal "collar" or ring was formed at their base, causing rigidity of the tissues round the ostium, which was slightly dilated. I found on taking sections from the insertion of the mole that chorionic villi with decidual cells were present.

Microscopical examination of the uterine end of the tube shows erosion of the epithelium almost over the whole surface of the folds, the lumen being patent.

CASE IV.

On Operation, right tube was found the thickness of the thumb, passing outwards and downwards to the haematocele sac, which was densely adherent all round; sac was ruptured and gave exit to old blood-clot. Left tube and ovary closed and sacculated.

Specimen. The tube is unruptured, and at its fimbriated end is the haematocele sac, the anterior wall of which is turned up to show its shaggy interior. The ovary is enlarged, the tube walls greatly hypertrophied; the abdominal ostium is patent surrounded by a peritoneal "collar." The mole was not found, but its attachment had been near the uterine end, on the wall of which I found chorionic villi with decidual cells. The uterine end of the tube was patent, and I found numerous glands in its walls, with several villi, when examined under the microscope. The haematocele sac contained laminated blood-clot, and the bursting of this sac was the cause of the symptoms which led patient to seek admission to the hospital. The right ovary contained a corpus luteum.

CASE V.

On Operation, right tube distended by mole ruptured during removal. The omentum was thickened and blood-stained; it was adherent to abdominal parities and to haematocele sac; the bowel also was adherent to the latter.

Tube was filled with firm blood-clot, protruding at the gaping abdominal ostium. Left appendages were normal.

Specimen. On opening into the tube a mole was found attached to its lower and posterior wall near the uterine end; it was pedunculated like a polypus, the upper part of the tube wall being cut away. The tube walls were greatly hypertrophied, plicae increased, and the ovary contained a fresh corpus luteum.

I found chorionic villi and decidual cells at the attachment of the mole; uterine end of tube was patent, and showed great increase in circular muscles and connective tissue, decidual cells being also found.

In this case patient's symptoms were all directed to the opposite side. This is not usual, but a similar case is mentioned by Taylor (*Extra-Uterine Pregnancy*, p. 32), in which a left tubal pregnancy caused a right iliac haematocele.

CASE VI.

On Operation, right tube dilated, omentum adherent over it; adhesions on upper surface very friable, but dense behind at junction of tube and uterus, where a coil of bowel was adherent posteriorly, dipping down into haematocele.

Specimen. Tube in section unruptured; a mole consisting of laminated blood-clot was attached to the upper and posterior part of the wall near the inner end of tube; walls of tube thinned at middle portion. No embryo found. Abdominal ostium patent. Plicae and fimbriae hypertrophied. Villi and decidual cells were found at inner part of tube.

CASE VII.

On Operation, a pool of serous fluid was found covering the contents of the abdomen immediately below the anterior wall; uterus and appendages completely enveloped in dense adhesions, out of which right appendages were raised, and found to contain pus. Chief mass in left side was composed of thin walled peritoneal cysts, packed round a spherical blood cyst about the size of an orange enclosed in a thick capsule

(ovary). Left tube presented a small blood cyst near its fimbriated extremity, which was the site of the pregnancy.

Specimen showed left pregnant tube with part of the haematocele sac. The site of the pregnancy was the middle third of the tube. The tube was enormously lengthened and thickened, but unruptured. Abdominal and uterine ends were patent.

CASE VIII.

On Operation, omentum bound down by greatly thickened border to upper surface of bladder and uterus by organising blood-clot; a large quantity of fluid blood in pelvis. Left tube lying in blood-clot.

Specimen. This case was very similar to Case VII. The left tube was greatly distended with blood-clot; the mole was near the uterine end, over which the tube walls were thinned (thickened, however, in the outer extremity). The blood was laminated in the mole, and no embryo was found. Abdominal ostium was patent, the fimbriae being surrounded by a "collar," and some of them withdrawn inside the tube-lumen. Examination near its uterine end showed a great increase of fibrous tissue, with erosion of the epithelium covering the surface of the plicae.

CASE IX.

On Operation, pelvis and lower abdomen were filled with blood-clot. Right tube enlarged. Left tube cystic, with blood-clot in outer part.

Specimen. The right tube contained a large fleshy mole near its ampullary end, attached to the lower part of the wall, where I discovered the presence of decidual cells. Chorionic villi also were present, but were extremely degenerated. Tube walls thin, but plicae increased. Abdominal ostium was patent. Uterine end of tube showed erosion of epithelium with numerous glandular interspaces with decidual cells.

In the majority of these nine cases of incomplete tubal abortion, the ovum had become attached near the uterine end; in only two was it near the ampullary end (Cases I. and IX.). In all of them the abdominal and uterine ostia were open,

although to the naked eye the latter often appeared occluded, and it was only when I made a microscopical examination that I found its lumen to be patent.

The tube walls in tubal abortion show the presence of compensatory hypertrophy more frequently than those which have ruptured. But it must be remembered that many cases which have been classed under tubal rupture have originally been those of incomplete tubal abortion, the tube being distended by haemorrhage, and afterwards becoming ruptured.

Rupture of the tube may occur very early in the pregnancy, or later on in those cases where the tube wall is more expansile; we, therefore, recognise the two forms as

1. Early Rupture.
2. Later Rupture.

Both of these may rupture with discharge of their contents into the peritoneal cavity or outside it, forming the extra and intra-peritoneal varieties.

1. *Early rupture* usually takes place before the sixth week, and is often the cause of very sudden and fatal haemorrhage. In these cases, I have observed great thinning of the tube walls, which condition probably accounts for the rupture, and may be due to some increase in the fibrous tissue of the wall at the expense of the more contractile or muscular tissue, as well as to the pressure of the growing ovum, which has perhaps become distended by haemorrhage. The event is generally hastened by such acts as straining during micturition and lifting of heavy weights. The rupture may be so small as to close up afterwards by the formation of adhesions, rupture with extrusion of its entire contents being delayed until a later period. But as a rule rupture takes place suddenly, and the haemorrhage is so severe that surgical aid is required immediately. When the tube wall bursts, the contents, including the embryo or mole, may become lost, especially if sudden, but if they are expelled by a gradual thinning, or so-called "erosion" of the tube walls, the membranes may remain intact, and thus the embryo will go on to a further stage of development. In *intra-peritoneal rupture* the tube wall bursts in the peritoneal cavity, and usually takes place

when the ovum is attached to the uterine or abdominal ends of the tube. When the ovum is attached to the middle zone of the tube it is more liable to burst in between the layers of the broad ligament, *i.e.* outside the peritoneum. The former condition is generally accepted as most usual, only a small portion of the tube being left uncovered by peritoneum; in nine out of twelve, however, of my cases of tubal rupture, the tube had burst in its middle third, the pregnancy being situated there. According to most authorities, the thinnest portion being at the attachment of the ovum, the ovum may or may not be expelled, and in some cases it remains blocking up the rent in the tube, while fresh haemorrhage is forming behind it; later on the pressure from the latter may expel it more forcibly. The blood may become encysted in the peritoneal cavity, forming a haematocele sac, adherent to surrounding organs, and roofed over by the protecting omentum. This may in its turn rupture, or become absorbed by slow degrees.

In extra peritoneal early rupture the contents of the tube with the resulting haemorrhage are discharged in between the layers of the broad ligament; the tubal space becomes by this means enlarged, and the embryo goes on increasing in size, or the haemorrhage is kept in check. In many cases the haemorrhagic tumour increases to a great size. In these cases the rupture is more often due to erosion of the tube walls, so that the embryo has a better chance for a further existence than in the intra-peritoneal form. The embryo still derives its nourishment from its original tubal attachment, and goes on growing surrounded by its amnion, which forms a lining to the haematocele sac. This is readily seen in many specimens which I have examined.

2. *Early rupture of the tube into the peritoneal cavity.* This takes place any time after the first month, but usually occurs about the third; it occurs earlier if the ovum is attached to the uterine end of the tube. The haemorrhage may not be so severe as in early rupture, but this depends upon the site of the placenta. As the tube contents increase in size, the walls become stretched and thinned, and are thus unable to bear the strain; one layer finally remains, which in its turn gives way,

and the gestation sac is extruded into the peritoneal or sub-peritoneal cavities. The resulting haemorrhage may set up a local peritonitis, which by its adhesions may limit the increase of the flow. Fresh haemorrhages occurring may break down these adhesions, and so we have fatal collapse and shock. As mentioned before, under tubal abortion, the haemorrhages may become deposited in layers, forming a laminated condition of the walls of the haematocele sac, and thus absorption may take place, or the sac may burst later on. If the rupture has taken place at the placental site, the haemorrhage is severe, owing to the rupture of the vessels. If it occurs away from the placenta in the thin tubal walls, the resulting haemorrhage is often slight in quantity. The placenta remains attached to the tube, while the foetus escapes upwards into the abdominal cavity, and the pregnancy may go on to full term, provided that the amniotic membrane has remained intact, as it acts as a protection against the peritoneum. This condition of abdominal pregnancy having its origin in the tube was first satisfactorily described by Taylor¹ in 1897, the amnion being in the form of a thin membrane or pellicle. In most cases the foetus after extrusion becomes buried in the resulting haemorrhage, and its blood supply being cut off, it either becomes completely absorbed, or decomposition with the formation of pus ensues. The placenta with the tube help to form the gestation sac, along with the haemorrhage which has become encysted. The foetus lying inside covered by its amnion increases in size until the sac bursts with the well-known signs and symptoms of intra-peritoneal haemorrhage. This gestation sac is adherent to surrounding organs. The placenta is situated below the foetus, as during rupture of the tube the foetus is generally discharged in an upward direction.

In extra-peritoneal later rupture of the tube, the pregnancy has generally begun at the middle portion of the tube; it generally expands, and passes in between the layers of the broad ligament, that is, into the connective tissue space below the peritoneum. When haemorrhage occurs a broad ligament haematoma may be formed, which by its pressure may stop

¹ *Extra Uterine Pregnancy*, p. 48.

the haemorrhage, and so become absorbed. When the rupture of this broad ligament in pregnancy takes place, the foetus escapes downwards, the placenta remaining attached above. The tube and placenta roof over the gestation sac, and the latter, owing to its high position, is a continuous source of danger from haemorrhage. The pregnancy displaces the peritoneum, raising it up from the surface of the uterus and bladder, and from the anterior and lateral abdominal walls, forming the so-called *sub-peritoneal variety*. At first it is confined to the pelvis, that is, *sub-peritoneo-pelvic*, but later on, as the haemorrhage increases, it invades the abdomen, and is then called *sub-peritoneo-abdominal*. This is when the pregnancy increases in an anterior direction. If it extends posteriorly it raises up the posterior fold of peritoneum from the sides and back of the uterus, from Douglas' pouch and rectum, and also from the posterior pelvic wall up to the sacral promontory. This is the limit in most cases, the anterior peritoneum being undisturbed.

On opening the abdomen, we at first think the pregnancy is everywhere covered by peritoneum, but if the hand is placed behind the haematocele sac the peritoneum is found reflected from above on to the mass. This form is called the *retro-peritoneal variety*, and has only been recognised for the last few years.

There is more room in this form for the growing foetus owing to the sac developing posterior to the tube and the foetus passing beneath the placenta. I have not discussed the advanced forms of extra-uterine pregnancy, as I wish to confine my remarks only to those cases which I can discuss and verify from my own personal experience. The table includes cases (x.-xxi.) of all the forms above mentioned, and are arranged as a means of illustration, their pathological anatomy being discussed fully in the text.

CASE X.

On Operation, whole lower abdomen filled with fresh blood-clot roofed over by omentum, which was adherent along its lower surfaces to anterior abdominal wall. Right tube stretched over haematocele and forming upper part of sac, was ruptured in middle third.

Specimen was completely disorganised by the surrounding haemorrhage, so that I could not make an illustration of it; the tube walls were very friable, and contained decidual cells. The contents had ruptured into the peritoneal cavity, with absorption of the embryo. This is an example of early rupture of the tube, the symptoms being sudden in onset and extremely severe.

CASE XI.

On Operation, left tube and lateral ligament greatly thickened, uterus pushed forwards and to right, bowels adherent to haematocele sac.

Specimen was entirely broken up, the tube being almost completely severed from the uterine wall by the suddenness of the rupture. Its walls contained round-celled tissue, with broken down chorionic villi and decidual cells about its middle third. The rupture had occurred before the fifth week. The embryo was absorbed by the blood-clot in the peritoneal cavity.

CASE XII.

On Operation, right tube was found to be ruptured; its walls were connected with the haematocele sac, which was filled with fresh blood-clot.

Specimen. Nothing could be made out except the ragged pieces of the tube walls, the whole being a mass of blood-clot. Rupture had taken place before the fifth week into the peritoneal cavity, the haemorrhage becoming encysted; this sac afterwards ruptured, giving rise to the more acute symptoms.

CASE XIII.

On Operation, haematocele, adherent all round to pelvic wall and posterior wall of uterus, ruptured during removal. Left tube enlarged.

Specimen shows tube sectioned down its centre; the whole tube was enlarged along the mesosalpinx, which was thickened; the mole was found distending the tube in its middle third, the abdominal ostium being closed. Rupture

had taken place into layers of broad ligament, and secondary rupture under the peritoneum. No embryo was found. Section of uterine end of tube shows atrophy of plicae, decidual cells, and glandular interspaces.

CASE XIV.

On Operation, a large mass of blood-clot and placental tissue was found connected with right tube. Foetus enclosed in membranes in centre of mass.

Specimen shows ruptured haematocele sac, foetus in lower part. The ruptured tube formed part of the upper wall of the sac along with the placenta, the tube having ruptured in its outer third, the other part of tube being intact, the walls thickened. The placenta was above, the partially macerated foetus lying below, attached by its cord to the placenta, the amniotic membrane lining the interior of the gestation sac. Rupture had taken place into the broad ligament, where the embryo had continued to grow until secondary rupture had taken place in below the peritoneum. Section of uterine end of tube shows hypertrophy of the mucous folds and the connective tissue elements.

CASE XV.

On Operation, left tube expanded out to form haematocele sac with its ovary were very adherent to surrounding parts.

Specimen. In lower part is ruptured tube, in upper is placenta with cord attached. Tube had ruptured in outer third in posterior and upper part; abdominal ostium fused in peritonitic adhesions was occluded. The tube walls were dilated, smooth, the plicae being stretched out into thin lines. Ovary was cystic, containing a corpus luteum, which had become partially transformed into a haemorrhagic cyst. The placenta was attached to the upper posterior surface of the tube, where rupture had taken place into the peritoneal cavity. Chorionic villi were taken from the placental tissue. No embryo was found. Section of uterine end of tube shows absence of plicae, with almost complete occlusion of the lumen.

CASE XVI.

On Operation, omentum adhering to brim of pelvis and to anterior abdominal wall, and surrounded by adherent bowel; immediately below this was foetus in an unruptured haematocele



FIG. 2.

sac, and surrounding it was broken down foetid blood-clot. Sac ruptured during removal. Patient died from septic peritonitis.

Specimen (see Fig. 2). In anterior is bladder with uterus and rectum behind, right tube completely disorganised, haematocele sac shown with part of anterior wall removed to show foetus in its interior. The foetus was about the twelfth week, and was below the placenta, surrounded by amnion, which lined the

gestation sac. Uterus was enlarged, and covered with adhesions. Section through its walls show groups of decidual cells, the part shown being near the mucosa. There was a great increase in fibrous tissue. Uterine end of tube shows great increase in muscular tissue, with patency of its lumen.

CASE XVII.

On Operation, a large quantity of fluid blood escaped; uterus was lying to right side. Large ruptured haematocele



FIG. 3.

sac on left side, from which a well-developed foetus was withdrawn. Placenta had extended upwards, and was

largely adherent to sigmoid; the cavity in the broad ligament extended deeply down into the floor of the pelvis below level of cervix. Total hysterectomy was performed for control of the haemorrhage.

Specimen (see Fig. 3). Uterus contains a twin pregnancy, transverse presentation. Right tube small and sacculated. Left tube almost completely torn from its attachment at the time of rupture; part of the haematocele sac with placenta is shown, with the foetus attached below by its cord. The pregnancy was advanced about four months. What had taken place was as follows:—The pregnancy had first ruptured in between the layers of the broad ligament, the walls of which had subsequently ruptured and the contents expelled below the peritoneum. The haematocele sac was formed by the dense adhesions which matted together the intestines in the upper part of the pelvis; and also by the placenta in its upper part. The interior of the sac was lined by amniotic membrane. This case is of great interest owing to the concurrent uterine pregnancy.

CASE XVIII.

On Operation, right tube distended in its outer part with blood-clot to about a diameter of 2 in. Below this was foetus; outer end of tube torn off from rest during removal. Placenta partly attached to enlarged tube. D.P. filled with blood-clot.

Specimen. In upper part is haematocele sac with placental tissue, below is foetus, which was of a slaty green colour with softened tissues. Rupture had taken place as in preceding case in between layers of broad ligament, then secondary rupture into the sub-peritoneal tissues. Section of the uterine end of the tube showed great increase in the connective tissue, the lumen being very small.

CASE XIX.

On Operation, left tube found ruptured and ovary contained several haemorrhagic cysts.

Specimen shows tube ruptured in outer third, and having chorionic villi attached. The tube was dilated, and adherent to and around ovary, its fimbriated end opening directly into the largest cyst of the ovary. Section near the uterine end of the tube shows patency of its lumen with erosion of the epithelium.

CASE XX.

On Operation, pelvic organs matted together with omentum adherent over and between them. Right appendages connected with a firm mass external to uterus and formed of blood-clot.

Specimen. In this specimen it was difficult to show the condition, as it was covered by adhesions. The tube was curved round the top of the haematocele sac, forming part of its roof. I slit this up transversely, and lifted the upper part of the tube, where it showed a rupture in its lower wall, the blood-clot protruding into its lumen. This blood-clot was continued down to the haematocele, the interior of which was lined with amnion; no embryo was found. The tube walls were hypertrophied, except at middle third, where they were thinned, rupture having taken place there into the broad ligament, with subsequent rupture in below the peritoneum; the haemorrhage extending behind, and pushing the peritoneum in front of it. The fimbriated end of the tube was patent, the epithelium being eroded; a "collar" of peritoneum was found round the bases of the fimbriae. At upper part of haematocele I found beautiful examples of chorionic villi. On making a thorough examination of the whole tube, I found a small rounded projection near its uterine end, about size of a pea, smooth, and attached to the lower and anterior part of the tube wall. This looked like a pedunculated mole. On section it contained firm blood-clot, on microscopical examination of which I found chorionic villi and decidual cells; in no other part of the tube could I find villi, so that those found belonged to the mole, and not to the other pregnancy described above. The question is,—Was this a previous or concurrent pregnancy, the mole remaining quiescent at an early stage, or was it a case of later impregnation of an ovum while pregnancy was present

in the outer part of the tube? The former seems the true explanation, as in the latter the ovum could not have passed down the tube from the ovary and become deposited near its uterine end while the outer part was blocked by the other pregnancy, nor is it likely that the ovum lay quiescent in the tube until fertilised. The history of the case shows that the patient had an attack of severe pain about a year before, but she got all right again. At that time there may have been a tubal pregnancy, with formation of a mole which became quiescent, giving rise to no other symptoms subsequently. These cases are rare.

CASE XXI.

On Operation, haematocele sac covered over anteriorly and above by uterus and distended right broad ligament, along which edge of omentum was attached. Sac adherent to posterior pelvic wall. Foetus in D.P.

Specimen. In upper part is dilated tube, ruptured in inner part, and surrounded by adhesions. Placenta has been removed from the tube, and is shown outside it with foetus attached below. Foetus about three months. Rupture occurred below the peritoneum, and pushed it forward from the posterior pelvic wall.

On looking over these cases, we find examples of all the forms of tubal rupture as before described. As examples of early rupture into the peritoneal cavity, we have cases X., XI., XII.: the tube in many of these early cases becomes so entirely disorganised as to be unfit for illustration. The most common form of tubal rupture in my experience is the broad ligament form—cases XIII., XIV., XVII., and XVIII. are examples of rupture having taken place into the broad ligament—then secondary rupture below the peritoneum, the haemorrhage taking an anterior direction, that is, the *sub-peritoneo-pelvic* form. In cases XX. and XXI. the haemorrhage extended in a more posterior direction—the *retro-peritoneo-pelvic* form. Rupture into the peritoneal cavity occurred in cases XIV., XV., and XVI. In most the attachment of the ovum had occurred in the middle third of the tube. In all the specimens that could be examined satis-

factorily, I found occlusion of the abdominal ostium, with the exception of case XX., where it was patent. The classification of these specimens is very difficult, as they present features similar to several forms, and they are often so completely changed by peritonitic adhesions, and by pressure, as to be almost unrecognisable as having any original connection with the Fallopian tube. It was only on repeated examinations by dissections, and by microscopic investigation of portions taken from along the whole tube wall, and also by serial sections by the paraffin method, that I was able to come to a conclusion as to their real form.

After this study of the tube wall, I observed the following changes due to the abnormal pregnancy :

The *serous* covering is in most changed and thickened by peritonitis. This is more apparent in the later months, when the surrounding pressure is greater. In many portions the entire interior of the tube wall was absent, only leaving the thickened serous covering. The walls become infiltrated with round cells, and in some I found spaces lined with cubical epithelium, as described by Webster, page 114. The *muscular* part of the tube wall is increased, especially the circular muscle fibres, as in those cases where compensatory hypertrophy has taken place. In later stages the muscle fibres become atrophied and scattered, the connective tissue increasing in quantity. Webster says the placental attachment to the tube wall renders it thicker. In my opinion the wall is thinner, and that it is there that rupture often takes place, as in case XV. The changes in the tube are less marked when rupture takes place early into the broad ligament. The *mucous* layer showed atrophy of the folds, with erosion of the epithelium, and in many the columnar had taken a more flattened or cubical form. I found decidual cells in many of my specimens, in some along the whole tube, in others only near the site of the pregnancy. These cells had no connection with the epithelial layer, as some believe. They are large, round, but usually oval in shape, containing several nuclei, and having no particular form of arrangement. The presence of these cells is denied by Bland Sutton. The capillaries in the tube walls are often enormously increased in size, especially in the deeper layers.

Case.	Age.	Married.	Children.	Youngest.	Last Menstrual Period.	Symptoms.
1	39	10 years	2 3 Abortions	1 year	12 weeks	5 weeks before sudden left iliac pain while at work, 2 days later "flooding," discharge ever since—5 days ago passed decidua
2	39	7 years	2 2 Abortions	1½ years	16 weeks	11 weeks ago severe "flooding," discharge present ever since. 2 weeks ago expulsion of decidua with labour pains
3	26	4 years	1 1 Abortion	1¼ years	3 months	2 months ago severe and sudden pain in hypogastrium, faintness. Haemorrhage present ever since
4	29	12 years	6	3 years	4 months	3 months ago severe "flooding," haemorrhage present ever since. Pain in right iliac region, no faintness
5	21	1 year	1	4 months	14 weeks	6 weeks ago severe pain in lower abdomen with haemorrhage, present to lesser degree ever since. Clots with "stringy" material
6	23	3 years	2	1 year	16 weeks	8 weeks ago severe pain in abdomen. Haemorrhage onset a week before, lasted 1 week. Returned for last 2 weeks
7	32	7 years	7	3½ years	14 weeks	10 weeks ago severe left iliac pain with haemorrhage, discharge ever since. "Flooding" days after onset

Signs.	P. V.	Condition of Uterus.	Condition at Operation.
Secretion in breasts. Hard, tender mass in left iliac region. Uterine haemorrhage	Cervix close to symphysis. Hard, irregular mass in D.P.	Enlarged	Left tubal abortion (incomplete) with mole containing foetus. Haematocele
Secretion in breasts. Flattened mass in hypogastric and left iliac regions. Haemorrhage	Cervix close to symphysis. Firm, irregular mass filling pelvis. Pulsation in left fornix	Enlarged, soft, sound passes 4" to right side	Left tubal abortion (incomplete) with foetus. Haematocele
Abdomen prominent in left iliac region. Large mass extending up to umbilicus. Haemorrhage	Cervix normal. D.P. filled by firm irregular mass. Rounded mass in right side	Anteflexed. Sound 3"	Left tubal abortion (incomplete). Haematocele. Right ovary cystic
Firm mass in hypogastric and right iliac regions. Not tender. Haemorrhage	Cervix close to symphysis. Firm round mass in D. P. and right side	Sinistroposed. Sound 3"	Right tube thickened passing into haematocele sac. Tubal abortion (incomplete)
Secretion in breasts. Fulness and tenderness in lower part. Haemorrhage	Cervix near pubis. Tense elastic swelling in D.P.	Enlarged and softened	Right tubal abortion (incomplete) Haematocele
Firm mass in lower abdomen. Extending more to right. Haemorrhage	Cervix close to symphysis. D.P. filled by firm elastic tumour. Pulsation in both fornices	Dextroflexed. Sound 3½"	Right tubal abortion (incomplete) with haematocele
Rounded mass in hypogastric and right iliac regions	Cervix normal. Rounded mass felt behind and to left	Anteflexed. Sound 3"	Right tubal incomplete abortion with pus

Case.	Age.	Married.	Children.	Youngest.	Last Menstrual Period.	Symptoms.
8	38	12 years	4 3 Abortions	1½ years	8 weeks	3 weeks ago sudden pain with hæmorrhage, fainted, sickness; . pain and hæmorrhage present till admission
9	30	12 years	4	3 years	2 months	3 weeks ago sudden pain with hæmorrhage, ceased for 2 days. Returned and present ever since
10	29	10 years	0	—	7 weeks	3 weeks ago severe abdominal pain in right side, faintness, collapse; 3 days later hæmorrhage, present till admission
11	33	10 years	1 1 Abortion 6½ years	8	11 weeks	6 weeks ago sudden pain in abdomen, vomiting and faintness. Hæmorrhage began a week before, lasting 4 weeks
12	31	9 years	6	1	3 months	2 months ago sudden hæmorrhage, 2 days later right iliac pain, present ever since
13	36	15 years	6 3 Abortions	3¼	4 months	3 weeks ago severe pain in lower abdomen, followed by a "flooding." Rigors and sweats
14	24	9 years	1	9	5½ months	7 weeks ago sudden pain in right iliac region, present ever since, sickness. Hæmorrhage immediately after, present ever since

Signs.	P. V.	Condition of Uterus.	Condition at Operation.
Abdomen tympanitic all over, but dull in hypogastrium. Haemorrhage	Right and posterior fornices filled by mass, irregularly solid and cystic. Pulsation	Enlarged	Pelvis filled with blood clot. Left tube distended. Haematocele
Fulness in hypogastrium. Dullness in lower abdomen, mass in right iliac region	Cervix close to symphysis. Fluid effusion filling D.P.	Sinistrotflexed	Right tube contained a fleshy mole
Great collapse, peritonitis. Firm mass in hypogastric and iliac regions. Haemorrhage	Cervix near pubis. Firm mass fills whole pelvis	Pressed forwards	Right tube ruptured. Fresh haematocele
Firm swelling in abdomen, extending up to umbilicus and to right iliac region	D.P. tensely distended with blood clot	Lies forwards and to right	Left tube ruptured. Pelvis filled with blood clot
Firm mass above pubis, irregular. Haemorrhage	Cervix behind symphysis. D.P. filled by firm uneven mass, not movable	Dextrotflexed, movable sound 2½"	Right tube ruptured. Haematocele sac
Rounded tumour in hypogastric and left iliac regions. Very tender	Left lateral and posterior fornices bulged down by a cystic swelling	Uterus anteposed Sound 3"	Left tube ruptured Haematocele
Firm mass filling whole lower abdomen and to right. Haemorrhage	Cervix close to symphysis. Firm, irregular, mass filling D.P.	Sound 4"	Ruptured right tube, fœtus, haematocele

Case.	Age.	Married.	Children.	Youngest.	Last Menstrual Period.	Symptoms.
15	36	11 years	1 1 Abortion 7 years	10	12 weeks	5 weeks ago hæmorrhage with severe pain, vomiting; 4 weeks ago passed a "flat triangular body"
16	33	16½ years	1	16	3 months	3 weeks ago sudden pain, faintness; hæmorrhage lasting 4 days. Pain and hæmorrhage at intervals since
17	30	?	4	1½	3 months	Since onset of pregnancy felt pain, with occasional hæmorrhage; faintness, collapse 4 days ago
18	38	13 years	8	7 months	6 months	3 months ago took a "flooding" lasting 6 weeks, ceased for 1 week. Present for 2 weeks before admission. Colicky pains
19	30	2 years	0	—	?	Previous ill-health, 6 months amenorrhoea 1 year ago. Incontinence of urine
20	31	3 years	3 Abortions	—	6 weeks	1 year ago severe pain with frequent micturition. 9 weeks ago sudden hæmorrhage and pain in right iliac region. Hæmorrhage till admission
21	27	1½ years	1	14 months	4 months	3 months ago severe right iliac pain, faintness and hæmorrhage. A month later hæmorrhage with clots, present ever since

Signs.	P. V.	Condition of Uterus.	Condition at Operation.
Tenderness and fulness in left iliac region. Haemorrhage	Tense elastic swelling in D.P. Pulsation in left fornix	Forward and to right	Left tube ruptured. Haematocele
Areolae round nipples, abdomen tender and resistant in both iliac regions	Cervix close to vulva. Fluid filling D.P.	Normal in size. Strongly ante-flexed	Right tube ruptured, foetus. Uterus enlarged
Collapsed. Abdomen distended, mass in middle line: to right is $4\frac{1}{2}$ months pregnant uterus. Dullness in both flanks, cystic boggy mass in left side	Soft mass in left side, very tender. D.P. free	Enlarged $4\frac{1}{2}$ months	Pregnant uterus Ruptured left tube
Secretion in left breast. Firm, resistant mass in hypogastrium, more to right side. Haemorrhage	Cervix enlarged and softened. Irregular, firm mass filling D.P.	Anteposed sinistrot flexed. Sound $3\frac{1}{2}$ "	Right tube ruptured, foetus, haematocele
Old cicatrices of previous abdominal operations	Left ovary enlarged and cystic. Right fornix free	Normal	Left tube ruptured, ovary cystic
Rounded mass in hypogastric and right iliac regions. Haemorrhage	Cervix low in vagina. Irregular, sensitive mass in right side of pelvis	Forwards. Sound 3"	Right tubal rupture. Haematocele
Firm, tender mass in right lower abdomen	Tense swelling filling pelvis, cervix close to symphysis	Anteposed	Right ruptured tube. Foetus, haematocele

DIAGNOSIS.

Before discussing the differential diagnosis of tubal gestation, it is necessary first to consider the symptoms and signs of this condition, as a knowledge of the latter is essential to the proper recognition of the condition. In my description I have adhered closely to my own personal experience of cases, and have only recorded the symptoms and signs as belonging to such.

In most cases the patient is within the child-bearing period of life; she has been going about her work in every way as if she were in perfect health, when suddenly, after perhaps some undue exertion, she feels a severe pain in lower part of abdomen: this pain is said to be like the stab of a knife or the shot of a gun. She becomes faint, and feels sick. The pain recurs, perhaps after a few weeks' amenorrhoea, or often at the time a normal menstrual period is due. The pain may have been preceded or followed by a haemorrhagic discharge which is often of the nature of a "flooding," and is due to some interference with the pregnancy, either by rupture of the tube or by dislocation of the embryo in incomplete tubal abortion: the source of the blood is the ruptured vessels in the tube. It also has its source in the uterus, when some of the decidual tissue has become separated. Cases of tubal pregnancy show no appreciable symptoms before rupture or abortion has taken place.

On looking at the patient what do we find? If the rupture of the tube has been sudden, the symptoms will be more severe than in tubal abortion: the face is blanched, with an anxious expression, lips livid, pupils dilated, and skin covered with a cold respiration; the respiration is sighing. In short, we have all the evidence of internal haemorrhage from rupture of some of the abdominal viscera. The pulse is at first slow, and then increases in rapidity, becoming difficult to count, the temperature is subnormal, and we see death approaching one who a few hours before was in the full vigour of life. If our experience has been limited, we pause and ask the cause of such a state. Our first thoughts turn to the ravages of poison, and we may even get the length

of applying the most likely antidote in our endeavours to aid the patient. We might think of rupture of the digestive viscera, or of cardiac failure. Seldom does the average general practitioner think of an abnormal pregnancy, and it is on his knowledge and promptness of action that the life of the patient depends. Death may take place unless surgical interference is resorted to. This portrait is drawn in the darkest colours, as these cases are not so common as those where haemorrhage takes place more gradually, namely in "tubal abortion" or in "tubal rupture" from gradual "erosion" of the tube. The patient has some slight pelvic pain, or if severe lasting only a short time, and complains of a haemorrhagic discharge which often comes on like ordinary menstruation, being, however, more copious and containing shreds or clots. She may have passed her period by a few weeks, and has had slight morning sickness. My experience is that the patient rarely, if ever, believes herself to be pregnant, as most of the symptoms are absent, although occasionally we do meet with typical cases. She stays in bed for a short time under the impression that she has had a "miscarriage"; her doctor is sent for, who probably treats her for such, and it is only on finding that the condition shows no improvement that he considers other factors in the diagnosis of the case. These attacks of haemorrhage generally recur several times, and the accompanying pain is often due to the local peritonitis which has been set up. In intra-ligamentary pregnancy the symptoms are usually slight when the tube ruptures, and they only become well marked when the walls of the ligament become so stretched as to rupture with consequent extrusion of their contents into the peritoneal cavity. What histories do we usually obtain from patients affected with tubal pregnancy? There is in most cases a history of *previous good health*, which is an important aid in diagnosis. *Previous sterility* is out of the question, as the majority of cases have occurred at the time when an ordinary pregnancy would have taken place: in one of my cases a child had been born three months before, and in another seven months.

Amenorrhoea is generally present, but not always; the patient is aware of a slight sanguineous discharge at intervals,

and complains usually of profuse leucorrhoea. (This symptom was present in all my cases, and is one that is scarcely mentioned by most writers.) Absence of amenorrhoea is no sign of normal pregnancy, as menstruation is possible for the first two months, that is to say until the decidua vera reflexa completely unite, which usually happens about the third month. The signs of pregnancy, such as morning sickness, secretion in the nipples, etc., may or may not be present.

Internal haemorrhage is very important, as it is seldom we see the patient before this occurs. The attacks may be single or repeated, the first occurring usually before the eighth week. The abdominal pain is severe and griping in character. There is generally some swelling and distention of the abdomen, but this symptom is more marked in tubal rupture than in tubal abortion.

Uterine haemorrhage is an important symptom, although not treated as such by most authorities. It was present in all my cases, and in most was the cause of the patient seeking admission to the hospital. This discharge comes on gradually, or as a "flooding," as it is described by most patients. It may contain blood-clot, with shreds of decidua tissue, or we may have a complete decidua cast. The blood is bright in colour, but afterwards becomes darker. According to Dr. Cullingworth the blood is always dark in colour, but in those cases which came under my notice the blood was bright red at first.

This uterine haemorrhage is a symptom indicative of some interference with the course of the pregnancy, and of danger to the life of the foetus. On examination of the patient we observe the following signs:—She may present the signs hitherto described under an acute attack; but as most of the cases which come under our notice have got over the acute stage, and are admitted to hospital complaining of haemorrhage and pain, it is to the milder form that we shall devote our attention—the patient is pale and anaemic, but otherwise healthy looking. Many cases have the appearance of good health, and when on subsequent operation we found the pelvis filled with blood-clot, it seemed to me that in no other condition do we have so great a variety in the facial expression.

The breasts are sometimes full, the nipple secreting, but often they appear quite normal, although some pigmentation is generally to be seen around the nipple; the tubercles of Montgomerie are more often seen described in the pages of the text-books than on the breast of the patient. The urine is often albuminous, probably due to pressure on the urinary tract, as it generally clears up after operation has been successfully performed.

The abdomen is enlarged and prominent in its lower part, and is distended if peritonitis has taken place; in many cases it is normal to inspection. The umbilicus may be depressed, especially if adhesions have formed between the anterior abdominal wall and haemorrhagic sac. On palpation a firm resistant mass is felt in lower part, tender to pressure, and usually extending more on one side than on the other, although not necessarily on the side in which the haemorrhage has its source; it is often hard and irregular, and even elastic in some places owing to areas of fresh haemorrhages. Seldom do we make out the foetal outlines, but often the fundus uteri can be made out as being more movable than the rest of the mass, which is usually fixed by surrounding adhesions. On percussion the abdomen is tympanitic, except where the mass is felt, and over it a changing note is heard, being deepest in the supra-pubic region. If the haemorrhage has been considerable the dulness may extend up the flanks, even to the costal margin, as in Case XVII. Occasionally the soufflé can be heard, but rarely the foetal heart sounds, as in most cases which come under our notice the foetus is dead.

P.V. The vulva and vagina are normal in the early stages, but have the port-wine colour of pregnancy later on when the pressure of the enlarging tumour has set up congestion. The tissues are very vascular, so that in most cases pulsating vessels are felt in either lateral fornix. The cervix is enlarged and softened, more especially in the early months: its position, principally in those cases of the retro-peritoneal variety, is close behind the symphysis pubis. So constant is this sign that I often relied upon it as one of the most certain signs of the condition. In some cases the cervix is pressed down by the tumour until it lies low in the vagina.

The uterus is enlarged and softened, and is slightly elongated. The enlargement is uniform, the characteristic antero-posterior thickening of uterine pregnancy being absent. It is often pressed forwards in the pelvis, being anteposed and flexed over to the side opposite to that in which the haemorrhage has taken place. Most writers describe the uterus as being retroposed, but the opposite condition has been my experience. The posterior part of the pelvis is generally filled by a tense mass continuous with that felt per abdomen, extending more to one lateral fornix than to the other. Sometimes when soft in consistence, the outlines of the foetus can be made out by the examining finger. This mass is found separated from the uterus by a distinct groove or by a ridge, but often the uterus lies embedded in the mass, and is difficult to differentiate from it. The vaginal walls are lax, and so obliterate the fornices. The sound—the passage of which should always be performed with extreme caution—usually passed beyond its normal length. There is usually tenderness and haemorrhage on examination. One point to notice in this condition is the gradual or even sometimes sudden enlargement of the tumour mass as evidenced by successive examinations, showing an increase of haemorrhage into the haematocoele sac or abdominal cavity.

DIFFERENTIAL DIAGNOSIS.

During the last few years our knowledge on the subject of tubal gestation has so increased that in most cases, where a thorough examination has been made, we can arrive at a fairly correct diagnosis. But cases are still to be found which show how limited one's knowledge is after all.

In the first place, we must find out the condition of the uterus and exclude normal pregnancy, then we should examine the appendages and exclude all other complications. If the examination is unsatisfactory, as in cases where there is great pain or extreme collapse, an anaesthetic will be of the utmost aid.

Tubal gestation is to be differentiated from :

- I. Simple Abortion.
- II. Early Uterine Pregnancy with Pelvic Tumour.
- III. Retroversion of Gravid Uterus.
- IV. Tumours of Ovary and Broad Ligament.
- V. Pyosalpinx.
- VI. Sub-Peritoneal Myoma.
- VII. Pelvic Haematocele and Pelvic Haematoma.
- VIII. Appendicitis.
- IX. Pelvic Cellulitis, Malignant Disease, etc.

1. *Simple Abortion.* In my opinion tubal gestation is almost invariably mistaken for uterine abortion. There is the period of amenorrhoea, with symptoms of pregnancy, followed by sudden bearing-down pains, with hæmorrhagic discharge containing blood-clot, coming on about the second or third month. There is perhaps the expulsion of the uterine decidua, which is looked upon as the complete expulsion of the products of conception. The patient is perhaps curetted, and this operation either leaves the symptoms unaltered, or it may bring on sudden hæmorrhage due to the manipulative interference. Our only safeguard after evacuation of the bladder and bowels is to make a careful bi-manual examination, and ascertain the exact condition of the uterus and the presence or absence of a tumour outside it. The difficulty of diagnosis is great when we get a rigid abdominal wall with a soft uterus and slightly patulous cervix, as sometimes occurs after recent hæmorrhage from the tube. If on careful curettage we examine the tissue microscopically, in tubal pregnancy decidual cells alone will be present; if decidual cells with chorionic villæ are found, the diagnosis is that of an intra-uterine pregnancy.

II. *Early Uterine Pregnancy with Pelvic Tumour.* This condition may very closely simulate tubal pregnancy. The uterus is softened and slightly enlarged with pulsation in the fornices, a cystic mass is felt bi-manually on one side of the uterus and filling Douglas' pouch, causing dulness to percussion per abdomen. There is the history of amenorrhoea, and perhaps breast signs, with morning sickness, etc.

In early intra-uterine pregnancy the uterus is softer and is thickened antero-posteriorly, the fundus is rounded and less defined. Thinning of lower uterine segment is felt per rectum. If previous chronic metritis has been present the condition is rendered more difficult.

On passing the sound in extra-uterine pregnancy the uterus is empty; if the interior of the uterus is thoroughly explored the presence of an intra-uterine pregnancy cannot be overlooked. There is, of course, the risk of inducing abortion in a uterine pregnancy, but it is the sacrifice of the lesser for the greater good, as the risk would be much greater if an extra-uterine pregnancy were allowed to go on. The presence or absence of amenorrhoea is no guide, as menstruation may occur, as mentioned above, during the first few months of normal pregnancy. If the patient be kept under observation, the uterus in intra-uterine pregnancy will be seen to increase in size. If uterine abortion has taken place, and if a pelvic tumour is present, the diagnosis can only be made by microscopical examination of the curetted tissue.

The following case shows how easily an intra-uterine pregnancy with tubal complications may simulate a tubal gestation.

Mrs. C., aet. 27, married two years, I.-para eighteen months ago. Menstrual normal in character.

History. Six weeks before admission patient took a severe pain in left iliac region, coming on suddenly, lasting for several days, and continued more or less until admission. She fainted two days before the onset of the pain, but has not fainted since. Previous to onset of pain she had $3\frac{1}{2}$ months' amenorrhoea, with other signs of pregnancy.

On examination, face flushed, temperature 100° Fahrenheit, pulse 106. Breasts secreting.

Abdomen protuberant, dulness reaching to 2 in. below umbilicus. Hypogastrium contains a sensitive and slightly movable tumour in lateral direction. P.V., cervix enlarged and softened, close behind symphysis. Tense, irregular, sensitive mass filling posterior part of pelvis, which presses on rectal lumen, presenting there a more cystic feeling. From examination and the history, the case seemed to be typical

of tubal gestation, with partial rupture and formation of haematocele. The faintness indicated an internal haemorrhage, and the acute pain two days later seemed to have its cause in intra-pelvic pressure and localised peritonitis. On the other hand the tumour was mobile, more like a $4\frac{1}{2}$ months' uterine pregnancy and there was no anaemia nor uterine haemorrhage. The possibility of an extra and intra-uterine pregnancy with some slight haemorrhage into D.P., the chief tumour mass being unruptured, was entertained. On abdominal section the uterus was found to be 4 months pregnant. The left tube was bent backwards and inwards, and adherent to bowel. The tube and ovary were found to be a mass of dark blood-clot, gangrenous in appearance, and adherent all round in D.P. Tube and ovary removed. Patient made a good recovery, and was afterwards delivered of a female child at term, the labour being normal in character. The tube contained 6 ounces of brownish fluid, its walls were $\frac{1}{4}$ in. thick. The ovary was size of a small orange, completely infiltrated with blood. Internal walls thick and leathery. Nothing could be made out on microscopical examination, although I examined sections from every part of the specimen. There is no record in any text-book of a case similar to this. There was no torsion of the tube, nothing but an acute bend at inner ends of tube and ovarian ligament. Vessels in walls were enormously increased in size.

The condition was due to an obstruction to the ovarian arteries and veins on the left side, caused by the bend. This probably took place at the attack of fainting. There may have been an old tubal condition present, and on the uterus rising out of the pelvis it may have caused a bend in the tube, which by its obstruction set up venous congestion, and thus we have the haemorrhage and subsequent necrosis. There may also have been a varicose condition of the tube present.

III. *Retroversion of Gravid Uterus.* Only in early months. In this condition there is great difficulty on micturition, owing to pressure from displaced cervix. There is sometimes retention with a distended bladder, also constipation. The cervix is close to symphysis, looking forwards and upwards,

and continuous with mass in D.P., that is fundus uteri. This condition of the fundus is found in some cases of early tubal abortion, but in most cases of tubal pregnancy it is lying forwards. In tubal pregnancy retention of urine is rare, and the uterus is separated from the mass, the cervix looking downwards and more to one side. In the latter there is the history of a previous acute attack, the uterus is empty, and is anterior to mass in D.P. The gravid uterus can usually be replaced, although attempts at such should never be performed until the position of the body of the uterus has been thoroughly made out.

IV. *Tumours of Ovaries and Broad Ligament.* These are sometimes difficult of differentiation, especially when small and when the ovarian disturbance has caused amenorrhoea.

If torsion of the pedicle of the tumour has taken place, the symptoms may closely resemble a ruptured tubal gestation, in so far as it causes a haematoma or haematocele—the symptoms of collapse also being present. The tumour, if ovarian, is usually separated from the uterus, but if intra-ligamentary it is very difficult, as the following case will show:—

Mrs. P., aet. 30, married eleven years, IV.-para, last six years ago, labour difficult owing to some pelvic obstruction.

History. Three weeks ago while lifting a heavy weight patient felt something give way in her right side, followed by very severe pain. Next day a profuse “flooding” came on with clots; this lasted for four days. Had another similar attack four days later, accompanied by vomiting and faintness. Six weeks’ amenorrhoea before onset of symptoms, but no symptoms of pregnancy were present.

Examination. Patient pale and anxious looking. Abdomen tender, slightly tympanitic.

P.V. Cervix lying forwards, and to the left. Pulsation in both fornices. Uterus enlarged in normal position, slightly boggy and lobulated. A mass the size of an orange is felt closely adherent to right lateral wall of uterus, and to posterior and right pelvic wall.

On reading the history of this case one would immediately think of a pregnant tube, the first rupture having taken place at the onset of the pain and haemorrhage. The uterus was

slightly enlarged, suggesting pregnancy. But there is also the history of pelvic obstruction at the last confinement, which would negative the former assumption, unless the pregnancy had been of old standing, when it would have been more likely to rupture at the time of labour.

On abdominal section, a mass was found between the layers of the right broad ligament, involving the ovary and tube, the latter being enlarged. On sectioning this mass it was found to contain pus, but no evidence of decidual cells or chorionic villi could be found, although I examined several portions under the microscope. The symptoms may have been due to a slight rupture of the cyst setting up pain, the haemorrhage being due to the involvement of a vessel together with the congestion of the surrounding parts. No rupture, however, could be found on examination of the cyst walls.

V. *Pyosalpinx* simulates tubal gestation closely, as in the double form the ovaries are often rendered inactive, and we have a period of amenorrhoea. There may be acute abdominal pain from a slight perforation, and if pus gets into the abdominal cavity, we have all the symptoms of collapse. On examination the pyosalpinx forms a tubal swelling, one side being more distended than the other, so that it feels almost one sided: there may be numerous adhesions simulating a haematocele. The points to note, however, in these cases are the previous history of ill-health, purulent leucorrhoeal discharge, with perhaps previous similar attacks: the signs of pregnancy being absent. Example:—

Mrs. R., aet. 36, married sixteen years, VI.-para, last two years.

History. Three weeks ago, after two months' amenorrhoea, while at work patient took a severe pain in the lower part of the abdomen and left iliac region; she felt faint and sick. Two days later had a similar attack, accompanied by rigors and uterine haemorrhage. Pain present ever since. Has had rigors and sweats, with vomiting on two previous occasions. Has had no suspicion of pregnancy.

Examination. Pale and anxious looking. Temperature $180^{\circ}2$, pulse 92. Secretion in both breasts.

Abdomen. A rounded firm mass felt in left iliac region, extending slightly to right of middle line.

P.V. Cervix enlarged, close behind symphysis. D.P. occupied by a rounded, irregular, doughy mass, slightly cystic. Uterus measures $2\frac{3}{4}$ in.

The diagnosis here was in favour of tubal gestation, and abdominal section was performed, when it was found that the mass was a large collection of pus, communicating with extremity of left tube and with cystic ovary; very adherent all round. Right tube and ovary enlarged and thickened.

We must always bear in mind the possibility of a pyosalpinx being the result of a tubal gestation which has become infected, as no doubt many originate as such.

VI. *Sub-Peritoneal Myoma.* Diagnosis easy, but a very adherent tubal pregnancy may be taken for a myoma. In myoma there is no history of pregnancy or of an acute attack.

VII. *Pelvic Haematocele or Haematoma.* These are very rare apart from ectopic, but may have their origin in :—

1. Regurgitation of menstrual fluid.
2. Varicose rupture in broad ligament.
3. Rupture of ovarian vessels.

There is the history and the absence of pregnancy to be considered in these. The following is an example of the third class :—

Mrs. S., aet. 35, married sixteen years, VI.-para, last eighteen months.

History. Eight weeks ago while undressing she suddenly took a severe pain in left iliac region, which caused her to faint. This was followed by a clear discharge, copious at first. She felt sick. Next day took a "flooding," the haemorrhage being present more or less ever since; it was bright red in colour, but did not contain clots or membrane. She had three subsequent attacks of faintness. Menstruation always regular, but was absent for five weeks before onset of symptoms; no signs of pregnancy were present. No secretion in breast.

Abdomen. A mass is felt filling lower part, more to right side.

P.V. Cervix close behind symphysis. Uterus anteflexed. D.P. filled by a large soft mass, fluctuant at one portion. On abdominal section under the diagnosis of a ruptured pregnant tube, we found the pelvis filled with about $1\frac{1}{2}$ pints of dark fluid blood with clots, numerous adhesions. Right tube normal, ovary distended with blood, and showing a rupture—the source of the haemorrhage. Other appendages normal. The question was, had we at last come upon an ovarian pregnancy? The symptoms were so typical of what one would imagine this condition to be; but nowhere could I find any evidence of it microscopically, although I made sections from all parts of the ovary and tube.

VIII. *Appendicitis*. In this condition we have the situation, history, and temperature to guide us; but there might be some difficulty in distinguishing between it and ruptured right tubal pregnancy, when in the former the uterus is so adherent that it cannot be made out, although in most cases the uterus can be diagnosed as free from the tumour. Appendicitis may be a complication of tubal pregnancy.

IX. *Pelvic Cellulitis, Malignant Disease, etc.* In these we have the history of inflammation, absence of pregnancy, and the difference on frequent vaginal examinations, the chief difficulty being at the menopause, where there are irregular haemorrhages, with perhaps colostrum in the breasts.

The diagnosis of such rare abnormalities as *pregnancy in a bi-cornute uterus*, or in a *rudimentary horn of a malformed uterus* need only be mentioned to be dismissed as being so rare. For the latter the treatment is similar, and for the former there is the presence of the vaginal septum, the furrow at the fundus, and the round ligament to the outer side of gestation sac.

Spurious pregnancy can be cleared up when an anaesthetic is used, although when occurring at the menopause, and complicated by a tumour of the appendages, it is sometimes difficult unless the patient is anaesthetised.

Tubal gestation has complications, such as:—

I. *Uterine and tubal together*, as in case XVII. Franklin, in the *British Medical Journal*, 1894, vol. i., page 1019, reports a case where patient was in labour, and owing to difficulty of

delivery Caesarean section was performed, and an extra-uterine foetus was found, the patient dying from haemorrhage, as so often happens in these cases.

II. *Uterine subsequent to tubal pregnancy* is rare, the foetus is usually calcified, and thus quiescent when the uterine takes place. Bozeman, in the *New York Medical Journal*, 1884, page 693, reports a case where after delivery a mass was found in the posterior fornix, it was incised, and the sac of an extra-uterine foetation was found, which had taken place some years before.

III. *Bi-lateral concurrent tubal pregnancy and repeated tubal pregnancy.* Examples of these are reported by Walter, *British Medical Journal*, 1892, October 1st; Edgar, *Tran. Glas. Obst. and Gyn. Society*, vol i., page 54; Haig Ferguson, *Edinburgh Medical Journal*, February 1899. The condition is more frequent on both sides. In my own case—see case XX.—the condition occurred on the same side.

REMARKS ON THE NATURE AND TREATMENT OF ASTHMA, WITH CASES.

By JAMES ADAM, M.A., M.D.

THE voluminous literature on asthma still leaves the nature of that disease an open question. The two recent pronouncements of Dr. Sidney Martin¹ and of Dr. Samuel Gee² show this. A summary of these will give a convenient *resumé* of the subject.

After excluding the dyspnoea due to emphysema and chronic bronchitis, cardiac and renal asthma, and pressure dyspnoea, Martin says spasmodic asthma is essentially due to nervous changes, and may be started by varying conditions, such as pollen (hay asthma), indigestion (peptic asthma), bronchitis (bronchitic asthma), infectious diseases affecting the lungs or nervous system (pneumonia or influenza), chronic disease of nose and throat ("not of so great importance as sometimes stated"). After remarking on the complicated relations of the respiratory centre (on the motor side through the thoracic, phrenic and vagus nerves, on the afferent side through the vagus and the sensory nerves of skin and upper respiratory tract), he says that this centre may be disordered by influences local or distant, and starting from the periphery by the sensory nerves of the upper respiratory tract or from the lungs themselves. He mentions the pathological theories: acute inflammation of bronchial tubes he dismisses at once. For non-inflammatory or vaso-motor swelling of the bronchial mucous membrane he says there is no evidence. He favours

¹ Martin, *Brit. Med. Journ.*, 1898, ii., p. 1861.

² Gee, *Brit. Med. Journ.*, 1899, i., p. 719.

the usual view that there is spasm of the unstriped muscular fibres of the smaller bronchial tubes, because it explains the sudden coming and going of the auscultatory phenomena, the patchy and rapid appearance and disappearance of the respiratory murmurs and rales at various parts of the chest. He holds that sufficient emphasis has not been laid on the nervous system, else how account for "cat asthma," "hay asthma," and even "bronchitic asthma," since it is not every case of bronchitis that develops asthma? Asthma, then, on Martin's view, is a neurosis due to the "condition of the central nervous system—a condition which cannot be defined categorically, but only expressed vaguely as that which is present in the individual predisposed to asthma,"—surely a lame pathology.

Contrast with this Dr. Gee's views, given three months later. Discussing the pathology of asthma, he says that "signs of bronchitis and pulmonary emphysema are always found; and although no one would call them the anatomical characteristics of asthma, yet the fact that they are constant demands that they should not be passed over or dismissed with the remark that they are mere complications." "Either bronchitis and emphysema constitute the morbid anatomy of asthma or it has no morbid anatomy. In either case we must reluctantly conclude that structural lesions cannot help us to a definition."

Of the nervous hypothesis for which Martin has just voted, Gee says: "One thing seems to be certain, namely, that if there be such a disease as bronchial spasm, pure and simple, it must be a very rare condition, because it is inadequate to explain the catarrh which manifestly attends most, if not all, asthmatic seizures." "The whole subject of bronchial spasm is involved in so much obscurity that we cannot admit more than that it perhaps takes a share in the production of asthma, but is seldom, if ever, the chief or only cause." He quotes with approval Robert Whytt, "that a true nervous or spasmodic asthma without any other fault in the lungs than uncommon delicacy or irritability of their nerves is a disease we seldom meet with"—a conclusion almost diametrically opposite to Martin's. Inspiratory spasm is not the

cause of the asthmatic paroxysm, Gee says, because the difficulty is often one of expiration; and finally he dismisses the nervous hypothesis. He adopts the hypothesis of "pneumonic asthma, for we have no third explanation before us." He then goes on, with a view to getting at the pathology of asthma, to consider certain closely related diseases, viz., (1) a peculiar form of bronchitis coming on suddenly, especially in children, attended by severe dyspnoea, but not by much fever, and passing off in a few days, with a great tendency to recur—this, he says, *is* asthma; (2) spasmodic croup, which is not laryngismus stridulus; (3) paroxysmal coryza; (4) paroxysmal bronchial flux; and (5) hay fever. By this breadth of view he gets nearer the pathology of asthma, and asks, in conclusion, "In what does the asthmatic tendency consist? Why are some persons prone to asthmatic catarrh, and others not? Why does the disposition run in families? What is the bond which in so many cases connects the several diatheses of asthma, eczema, and gout?" An attempt to show that Dr. Gee's view, which seems nearly the reverse of Dr. Martin's, is merely the obverse of the shield; and in some measure to answer his concluding questions, was the origin of this paper. It is based on facts which had long been known to me, but to which attention has been drawn only recently, if at all.

First, I wish, as a general practitioner, and notwithstanding Gee and Martin, and perhaps Sir Felix Semon,¹ to the contrary, to emphasise the importance of nasal disease in asthma. It was Herck, of Freiburg, who, in 1844, first insisted on this. His view was carried into excess, and is now suffering from the usual reaction. The true respiratory orifice, the orifice of the respiratory cavity, is the nose. Like other orifices, it is the most sensitive area of, and when diseased is liable to set up spasm in, the correlated viscus or viscera. (Cf. irritation at neck of bladder, fissure of anus.) Its widespread nervous connections are illustrated by the analogy which Gowers has pointed out between a paroxysm of sneezing and tetanus. Both involve contraction of muscles in head, neck, trunk, and limbs. The nasal mucous membrane derives general sensation

¹ Semon, *Clinical Journal*, 1900, i., p. 24.

through the fifth nerve, which is also its vaso-motor nerve; it is largely a specialised erectile tissue requiring constant vaso-motor control. As Bosworth¹ says, "The nasal passages contain an exceedingly important, perhaps the most important, and certainly the most intricate apparatus connected with the function of respiration, of the whole respiratory tract, and one on whose normal functional activity depends the integrity of the whole of the mucous membrane of the respiratory tract below." Not only does the nose purify the inspired air, but it supplies or should supply most of the moisture and heat of the *expired* air, the function of the lungs being the interchange of gases and not the heating or moistening of them. Hence nasal disease is apt to be associated with asthma in three ways: (1) Reflexly, through the nervous system. The sensitive respiratory orifice is bound to send impulses to all parts of the respiratory tract. (2) Where the nasal disease is obstructive, the onus of warming, moistening, and purifying the inspired air is thrown on the bronchi and lungs, which are thus being constantly irritated and made liable to more or less chronic inflammation. (3) A diseased area is always a nidus or focus of inflammation, liable to attract disease to itself and to spread it elsewhere.

These physiological and pathological considerations are supported statistically. Of the appended 25 cases of asthma, which are all I have had the opportunity of thoroughly examining and treating, only two had no nasal disease; *i.e.* in 90 per cent. of unselected cases of a spasmodic disease which affects the respiratory tract and in which it is admitted the nervous system must play some, and probably an important part, the most sensitive area of that tract was not normal. It is not suggested that the nasal disease is an equally important factor in all the cases (in Cases 6, 22, 24, it is of quite minor importance); still less is it suggested that curing the nasal disease will cure the asthma. But the importance of nasal disease in many cases can be variously demonstrated. (1) Curing the nasal disease sometimes cures the asthma. The first case that made me realise the importance of nasal disease in asthma was a miner, aged about 30, who came to

¹ Bosworth, *Diseases of Nose and Throat*, 1889, i., p. 89.

me eight years ago. For three nights he had not slept owing to severe asthma. As he seemed able to breathe easily enough with his mouth shut, I was about to prescribe for him, thinking there was no need to examine his nose. However, I thought it wiser to make sure, and to my great surprise discovered in the left nostril a single, somewhat fibrous polypus, about the size of a small cherry. This I snared, and the patient, so far as I could learn, had no more asthma. This, however, is not a common result. (2) Again, *in an asthmatic patient*, nasal treatment may produce or allay an attack of asthma. A nasal operation, especially with the cauter, by producing reactive inflammation, often in a day or two results in a severe attack (Cases 2, 4, 8, 12). On the other hand, where an attack is accompanied by engorged turbinals the effect of applying to the turbinals a 15 per cent. or 20 per cent. solution of cocain followed by one of suprarenal extract is often marvellous. In five or ten minutes they are blanched and shrunken, the dyspnoea and wheezing rales have gone, the cardiac and hepatic dulnesses, previously reduced by over-distended lungs, may have again increased (Cases 2, 4, 16). This effect varies in different cases, and at different times in the same case, depends much on the thoroughness of the application and the nature of the case, is greatest where the turbinals are simply engorged and lying against the septum, is least where there is actual inflammation. Engorged tissue is shrunk and white; inflamed tissue stands out red and defined. The more or less complete cessation of cooing rales is probably largely due to the quiet breathing, as compared with the previous laboured efforts of the patient. Moist rales are less if at all affected; reduction of cardiac or hepatic dulness by emphysematous lung is not altered. But the alteration in the patient's distress is often gratifying. This action seems to be a reflex one produced by the local effect of the drugs on the nose. It is not due to the vaso-constrictor action of suprarenal extract absorbed into the system, for (1) it is too prompt and (2) I have frequently used suprarenal solution to staunch bleeding from skin incisions in small operations, which it does promptly and for some time.

In connection with the question of nasal disease in asthma,

it is well to remember that asthma and adenoids may complicate each other, but that the dyspnoea of adenoids and that of asthma are different, the former being nasal both in seat and origin, the latter being pulmonary in seat, though it may be of nasal origin; that while both may occur in young children and are worst at night, and while the adenoids may have much to do with the production of nasal disease, of bronchitis, and, through them, of asthma, yet removing the adenoids will not cure the asthma. This was well exemplified in Case 15. This child, aged 5, suffering from asthma, bronchitis, nasal obstruction, and adenoids, had its adenoids and nasal hypertrophy removed by Dr. Walker Downie with great improvement in its general health and mitigation, but not cure, of its asthma. On the same day Dr. Downie removed very large adenoid growths from a case that looked very similar—a child who had bronchitis and great dyspnoea during sleep, but no asthma and no great intra-nasal hypertrophy, and the dyspnoea entirely ceased. Again, when in adults the nasal cavities are full of large growths the patient is apt to have dyspnoea, because, being forced to use the mouth for breathing as well as for chewing and speaking, these two latter acts, especially chewing, prevent the patient getting sufficient breath, and every now and then he feels like to choke. And in some patients this is worst at night. Remove the growths and you cure the dyspnoea just as with adenoids. *Doubtless such cases sometimes get the reputation of being cured cases of asthma*, and Case 19 may be amongst them.

Considering the appended cases still further, we find sometimes hypertrophy of the lingual and faucial tonsils (Cases 19 and 22). The lingual tonsil is apt to be neglected, and when hypertrophied to cause constant irritating cough. Though it has probably little to do with asthma, it ought always to be examined and, if necessary, treated. Case 20 seemed most benefited by treatment of lingual tonsil, though the nose was also attended to. Painting base of tongue with cocain solution seemed to ease patient's attacks of asthma.

In the two cases which showed no nasal disease—a mother and daughter—bronchitis had been more or less persistent

for years before the asthma began, so that the bronchial tubes would be more or less irritable—the very parts that on one theory are the active cause of the asthmatic spasm. In the cases recorded, then, we find a lesion, a vulnerable spot, in the respiratory tract in 100 per cent.

One word on those peculiar cases called “cat” asthma, in which the presence of a cat or other animal causes asthma. That this influence is conveyed through the sense of smell and acts through the nose is undoubted. There is not one of the senses in which acuteness varies more amongst different individuals than that of smell. This peculiar “cat” asthma of itself speaks for the influence of the nose on asthma in general, as also does the irritable condition of the nose found in cases of hay fever, which go on to “hay asthma.”

Though 100 per cent. of cases of asthma have a lesion, a source of irritation in the respiratory tract, it is not every case of nasal or bronchial disease that develops asthma. This brings us to consider another striking feature of the cases recorded—one connected with the essential pathology of asthma, yet one which has not, I think, been pointed out before, though Bristowe¹ refers to it in a general way—I mean what, for want of a better term, may be called the “week-end” periodicity of asthma. Ten of these cases were men. In eight of these the asthmatic attacks occurred more or less regularly on Mondays, sometimes beginning on Sundays, sometimes being worst on Tuesdays (Cases 4 and 12). The two men not showing this “week-end” periodicity were Case 1, a miner, who came to me before I realised this peculiarity, and who had not long had asthma; and Case 6, who was an architect, having regular meals all week and no great bodily labour. The first time I became aware of this periodicity was in 1893, in Case 2. The regularity with which he came to me on Mondays was remarkable. Yet this periodicity will be missed unless specially inquired for. I have since found that most working men with asthma have their attacks on Mondays. They work five and a half or six days a week; on Sundays have less exercise and more food—food, too, of a different kind and at different hours. A woman’s work, on

¹ Bristowe, *Theory and Pract. of Med.*, 5th edition, p. 473.

the other hand, varies less than a man's as between Sundays and week days; and asthma in women seldom manifests this "week-end" periodicity. In Case 21, however, the periodicity tends to show itself. She is a young woman who works hard all week at a tailor's sewing machine, and rests on Sunday. Further, as the boy (Case 24) has grown up, and especially since he has gone to work, he has had with great regularity to give up work on Mondays on account of asthma. Case 4 was off work for a considerable time owing to asthma and operative procedure on his nose, but whenever he resumed work his asthma stuck to Mondays in very striking fashion. Still further, since Case 8 has taken to doing a little cycling on Sundays he has been rewarded—for his breach of the Fourth Commandment, as it is in Scotland—by some improvement in his asthma. He has sometimes to work on Sundays, and then never has asthma on the following day.

Now this peculiarity seemed to me to point very plainly to some error of metabolism, or of elimination, less probably of absorption, consequent on the amount of food not being cut down on Sundays in proportion to the amount of exercise, but rather increased.

Another periodicity is well known, viz., that asthma is usually worst at night or just about the time of waking.

Both periodicities, but especially the "week-end" one, point to a toxic condition of the blood. This should express itself in the urine, more emphatically in the urine during or immediately following attacks of night asthma or "week-end" asthma. If the error be one only of elimination, its expression is more likely to be found in urine towards the end of or immediately after than during an attack of periodic asthma. In short, we may have to blame a toxin, or that old scapegoat, uric acid.

A series of observations was made with regard to uric acid. The urine was separated into day and night portions of twelve hours each, and these examined separately. Difficulty and delay always attend such observations in private practice, but the results, though not perhaps absolutely accurate, are relatively accurate enough to allow conclusions to be drawn.

To aid this a fairly large number of observations was made. Uric acid was estimated by Rosenheim and Tunnicliffe's¹ modification of Gowland Hopkins' process; urea by the Doremus ureometer, which gives only approximate results, urea tending to be too high.

Table I. shows urea and uric acid of two men who consider themselves in sound health—(1) is the average of six observations on one man by myself on three different days; (2) gives the results got by Dr. Carstairs Douglas from the same man on another day; (3) gives results from another man with nasal polypi, but no asthma. The results are all much alike, except

TABLE I.

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
1. Day, - - Night, - -	1051 549	14·2 8·48	·148 ·075	{ Average of observations on 3 days' urine of a healthy man by myself.
2. Day, - - Night, - -	1420 497	14·2 8·449	·478 ·084	
3. Day, - - Night, - -	597 398	13·129 12·332	·158 ·106	{ Man with nasal polypi, but no asthma.

TABLE II.

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
Jan. 25th 8 a.m.—8 p.m.	511	14·324	·233	Slight asthma.
Jan. 26th 8 p.m.—8 a.m.	313	10·004	·082	Severe asthma.
One day in March				
Day,	682	17·734	·455	Severe asthma.
Night,	256	7·418	·283	Asthma less.
Average—				
Day,	596	16·029	·344	
Night,	284	8·711	·182	

¹ Rosenheim and Tunnicliffe, *Brit. Med. Journ.*, 1898, i., p. 365.

that the uric acid of the day period in (2), when the man was working much harder, is rather high, while it is rather low in the night period of both (1 and 2). The quantity of urine in the day period of (2) is thrice that of the night, and thus exaggerates the difference in uric acid of the two periods; in (1) the quantity of day urine is only twice that of night urine. Urea is much the same in all; in no case is urea or uric acid greater during the night than during the day.

Table II., put below I. for comparison, shows results from Case 2. The total uric acid tends to be high, but is much less on the night of "severe" asthma than on the night when asthma is "less"; the reverse holds for the uric acid of the daytime. Both urea and uric acid are up on the day period of "severe asthma." Note the diminished quantity of urine, especially at night, in Table II. as compared with I. On one night of severe asthma (twelve hours), not referred to in the table, this patient passed no urine.

Tables III. and IV. are from Case 4. III. was during September when asthma occurred every night. Uric acid is high, higher sometimes even at night than during the day; urea also tends to be higher at night than during the day. In this connection, note the averages. IV. was during October when asthma was less troublesome. On the whole, urea is less; uric acid much less, especially at night. (Contrast the averages of Table IV. with those of Table III.) But there is no regularity in the rise and fall of uric acid with the presence or absence of asthma, *e.g.* there was asthma on night of October 27th, urea gm. 9.09; uric acid, gm. .073; on night of November 1st, without asthma, urea is again gm. 9.845, but uric acid is gm. .203. If periods of twelve hours during which there was asthma, and similar periods asthma-free, be taken, we get for the asthma periods an average of 10 gm. urea and .256 gm. uric acid, for the asthma-free periods 8.8 gm. urea, .284 gm. uric acid. So that uric acid does not tie itself regularly to asthma. It is possible, but not probable, that uric acid would show more regular excess if the urine taken were strictly limited to the duration of the asthmatic attack instead of to twelve-hour periods during which asthma occurred. This idea is supported by Tables V. and VI. from

TABLE III.

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
September.				
14th, 8 p.m. to } 15th, 8 a.m. }	469	11·253	·6290	Asthma more or less throughout. Worst at night and on waking.
15th, 8 a.m. to } 8 p.m. }	426	9·804	·3317	
15th, 8 p.m. to } 16th, 8 a.m. }	469	11·253	·3224	
16th, 8 a.m. to } 8 p.m. }	554	11·636	·3766	
16th, 8 p.m. to } 17th, 8 a.m. }	511	14·8335	·4148	
17th, 8 a.m. to } 8 p.m. }	597	15·5168	·2291	
Averages—Day, Night,	— —	12·319 13·043	·3125 ·455	

TABLE IV. (Same Patient as III.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
October.				
27th, 8 p.m. to } 28th, 8 a.m. }	284	9·0944	·0731	Asthma.
28th, 8 a.m. to } 8 p.m. }	455	11·822	·2759	Asthma half day.
28th, 8 p.m. to } 29th, 8 a.m. }	341	8·525	·1931	No asthma.
29th, 8 a.m. to } 8 p.m. }	398	9·1494	·479	Asthma.
30th, 8 p.m. to } 31st, 8 a.m. }	398	9·945	·2996	Asthma.
31st, 8 a.m. to } 8 p.m. }	256	8·1824	·4587	No asthma.
Nov. 1st, 8 p.m. to } 2nd, 8 a.m. }	199	9·845	·2035	No asthma.
Averages—Day, Night,	— —	9·718 9·427	·4044 ·1924	

same patient as Tables III. and IV. V. shows uric acid excreted at different periods of twenty-four hours asthma-free; VI. gives urea and uric acid of two corresponding periods with asthma the whole time. The total uric acid of the latter two periods, though covering only ten hours, is twice as great as that of the whole twenty-four hours asthma-free.

TABLE V. (Same Patient.)

	Urine in C.C.'s	Uric Acid in gms.	REMARKS.
May.			
4th, 7 p.m. to 11 p.m.	171	·094	No asthma.
4th, 11 p.m. to 5th, { 5 a.m.	142	·0154	No asthma.
5th, 5 a.m. to 10 a.m.	142	·0356	No asthma.
5th, 10 a.m. to 7 p.m.	317	·148	No asthma.
		·2929=	Total $H_2\bar{U}$ for 24 hours.

TABLE VI. (Same Patient.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
May.				
13th, 7 p.m. to 11 p.m.	114	4·317	·3042	Asthma.
13th, 11 p.m. to 5 { a.m. 14th	142	5·346	·2034	Asthma.
			·5076=	Total $H_2\bar{U}$ for 10 hours.

Table VII. gives a similar result. The case (22) is a lady suffering from malnutrition, her weight (in walking dress) is only $6\frac{1}{2}$ stone. For nine hours during which there was asthma, the total uric acid is gm. 0·3848 for the succeeding fourteen asthma-free hours, the total is only gm. 0·1045, say one-third. Urea is much the same for the two periods. With this contrast Table VIII., from a woman weighing 10 stone, who suffered from chronic bronchitis, marked emphysema, and probably some bronchiectasis, but who *had*

TABLE VII.
(Female: Weight $6\frac{1}{2}$ stone.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
9 hours— 5 p.m.—10 p.m.	170	3·9215	·1592	Asthma { Total— Urea = 5·797 H ₂ \bar{U} = ·3848
10 p.m.—2 a.m.	170	1·8755	·2256	
14 hours— 2 a.m.—7 p.m.	170	2·387	·0387	No asthma { Total— Urea = 5·57 H ₂ \bar{U} = ·1045
7 a.m.—4 p.m.	114	3·183	·0658	

TABLE VIII.

(Female; Bronchitis, emphysema, normal nose, no asthma. Weight, 10 stone.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
July.				
29th, 8 p.m. to } 30th, 8 a.m.	738	9·9684	·1038	Averages— Urea—Day, 7·966 Night, 10·366 H ₂ \bar{U} —Day, ·1692 Night, ·1413
30th, 8 a.m. to } Day 8 p.m.	483	7·966	·1676	
30th, 8 p.m. to } 31st, 8 a.m.	511	11·7576	·0811	
31st, 8 a.m. to } Day 8 p.m.	426	8·52	·1814	
31st, 8 p.m. to Aug. } 1st, 8 a.m.	426	9·372	·239	
Aug. 1st, 8 a.m. } Day to 8 p.m.	256	7·4142	·1587	

TABLE IX.

(Female; diseased nose, asthma. Weight, 9 stone.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
February—Night,	568	13·0732	·2996	
Day,	540	12·9595	·3248	
Night,	313	8·4407	·203	
Day,	426	9·8049	·2575	

neither nasal disease nor asthma. Uric acid is less, the average for twenty-four hours being gm. 0·3105 as contrasted with 0·489 for twenty-three hours of the asthmatic patient; or 0·1164 for nine hours of the asthma-free patient as contrasted with 0·3848 for nine hours of asthma in the other.

Table IX. is from Case 10, an asthmatic lady weighing about 8 stone, who happened to have little or no asthma during the time represented. Here again uric acid is high. This lady has to my knowledge long passed excess of uric acid.

Table X. shows somewhat irregular results in an asthmatic man (Case 20). Uric acid is high during the "severe asthma" period as compared with other night periods, except the first, which was asthma-free, and has the largest excretion of all.

TABLE X.
(Man. Weight, about 12 stone.)

	Urine in C.C.'s	Urea in gms.	Uric Acid in gms.	REMARKS.
Sept.—Night (12 hrs.)	511	16·368	·3340	No asthma.
Day (12 hrs.)	583	12·558	·1768	No asthma.
Night (12 hrs.)	539	10·241	·1137	Asthma.
Day (7 to 8 p.m.) }	284	{ 5·968 (8·952)	{ ·1097 (·1645)	Asthma in evening after a hearty dinner. N.B.—This is only an 8-hour period. (A 12- hour period at this rate would give):—
Night (12 hrs.)	398	9·1517	·2992	

To sum up, the results here stated, and others which I have, go to show that asthmatic patients have a relatively high excretion of uric acid even when they have no asthma. It tends to be highest during periods of asthma, and may even be higher during the night than during the day, thus reversing the normal. This rule is not universal, for rarely (see Table II.) uric acid is least when asthma is worst, as if it were being held up. But it is significant that uric acid in all the cases of *asthma*

in which its estimation was undertaken, was found to be high, and there was more or less disease of the respiratory tract, whereas in each of the two cases where there was marked chronic disease in that tract without asthma, uric acid was not high. We can thus see that there is a close connection between asthma and excess of uric acid, and this answers, to some extent, Dr. Gee's question, "What is the bond which in so many cases connects the several diatheses with asthma, eczema, and gout?"¹

While working at the relationship between uric acid and asthma, I became acquainted with Bouchard's work, *Auto-intoxication in Disease*,² and I think that there, though he does not show the relationship I wish to point out, may be found the true explanation of the asthmatic paroxysm. Speaking of normal conditions, he says (p. 41): "The urines of the day period do not only differ from the urines of sleep by a toxicity twice greater, but the toxicity of these two urines presents differences of a qualitative character. The urines of sleep are always markedly convulsive." (This would explain "night" asthma, which is apt to be worse towards morning.) "Those of the day period are very little or not at all convulsive, but they produce narcosis. . . . What is certain is that during the day the body forms a substance which when accumulated would induce sleep, and that during sleep it elaborates, instead of this narcotic substance, a convulsive substance, which, when accumulated, could produce muscular twitchings and induce waking." Again (p. 43), "The toxicity of the urine of sleep being only half of the toxicity of the urine secreted during an equal period of the day, we might think that the urine of repose ought to be less toxic than the urine of muscular effort. *But it is the contrary that is true.* One day of great muscular activity spent in the open air

¹ After I had begun these observations, Dr. Armstrong, of Buxton, wrote a letter to *Brit. Med. Journal* (3rd June, 1899, p. 1331), expressing surprise that at a recent meeting of the Laryngological Society no one but Dr. Dundas Grant had remarked on the connection often observed between asthma, eczema, and gout. The subject of discussion was Asthma. To my friend Mr. R. M. Lawson I wish to express my hearty thanks for much help in the estimations of urea and uric acid.

² Bouchard (Oliver's translation).

in the country diminishes the toxicity of the 24 hours by one-third, and on that day the toxicity does not diminish only during the time devoted to muscular exercise. The toxicity which diminishes during work remains less during the repose which follows this work and *during the sleep which succeeds this day of muscular activity.*" Bouchard proves his statements by experiments. If then we regard the urine as an expression of the blood, his remarks are exactly what we should have expected him to deduce from observations on "week-end" asthma. They amount to this: Diminish exercise and you increase the toxicity of urine—you increase the narcotic effect of the day urine, the convulsive effect of the night urine. If asthma is due to such a toxic agent, we should expect it to be worse at night, and especially on the night following a quiet day; we should expect it to be worse on Sunday night or Monday morning. And I have shown that it is so. Bouchard shows that the convulsive substances obtained from urine are insoluble in alcohol, and are two, an alkaloid and potash. This alkaloid "is found in less quantity in the urine of the day period than is the narcotic material, but it is of less physiological activity." His previous remarks leave one to infer that it is in greater quantity in the night urine.

Further, my records of asthma show gastro-intestinal disturbance in all the severe cases. (It was present in Cases 2, 4, 7, 9, 10, 12, 17, 20, 22, and probably others.) Many of them had to avoid certain articles of diet in order to avoid asthma. In Case 7 a very bad attack occurred after a "feed" of fruit and beans. And the alterations of diet, as to time, kind, and quantity, on Sundays also tend to cause gastric derangement, though I do not think to such an extent or with such regularity as to be the sole cause of "week-end asthma." Patients suffer too much not to mend their diet in this way, and do alter it where they find it advisable. The periodicity of the asthma is much too regular for this to be the sole cause of it. But it would be an adjuvant to other toxic conditions, since, as Bouchard shows, part of the toxicity of urine is of intestinal origin (p. 101). And again (p. 140), he says, "If I suppress intestinal fermen-

tations I cause the toxicity of urine to diminish: I cause it to diminish but not to disappear, since I only suppress one of the natural sources of its intoxicity."

It was thought possible that indicanuria might show some relationship to asthma, especially if asthma were due to an error in absorption. A number of observations, therefore, were carried out on Herschell's¹ comparative method, modified to suit 12-hour periods. The urines passed during or immediately after asthma periods were compared with the urines of asthma-free period. An indican reaction was not at all constant, was often very slight, and was worst in a case of "bilious vomiting" unconnected with asthma, and taken for comparison. The results are not worth detailing, and showed no constant relationship between indicanuria and asthma. Therefore it is probable that any poison absorbed from stomach or intestine will only be an adjuvant to that which is most active in asthma, and which probably results from faulty metabolism or defective elimination, or both. The proof of this hypothesis of the toxic nature of asthma would be the injection, into animals, of urine passed during or immediately after an attack of asthma and comparing the results with the injection of urine of asthma-free periods. One would expect convulsions to be produced by much smaller doses of the former than of the latter. Such a proof it is impossible to carry on in a country town. But I can adduce clinical evidence of the truth of the toxic hypothesis.

Many of the appended cases showed symptoms referable to toxins. Several of them showed urticaria. Case 6 had only one attack of it; but he had also, at a time when his asthma was specially frequent, an attack of Raynaud's disease or "local asphyxia"—a very interesting complication, to which I shall again refer, and one which is held by some to be due to a toxin. Case 10 has frequent urticaria; in Case 14 it is chronic, and occurs nearly every night; in Case 7 an attack of asthma was usually signalled beforehand by tingling and irritability of the skin, which became dry and harsh. This case and Case 9 also used to develop petechiae of the skin during severe asthma. Many writers have pointed out

¹ Herschell, *Brit. Med. Journ.*, 1899, ii., p. 1257.

the toxic origin of urticaria,¹ and Bouchard refers (p. 164) to the connection of cases of dilated stomach with paroxysmal dyspnoea recalling asthma, as well as with sneezing and coryza. Many cases are on record of erythematous rashes following enemata due to the washing of toxic substances from the faeces and their absorption by the bowel. The following remarkable case of the "acute circumscribed cutaneous oedema" of Quincke, or subcutaneous "non-itching" urticaria, was clearly due to a toxin. It is remarkable because, like some cases of asthma, it occurred on four successive Sundays, and only on Sundays.

G. F., aged 17, a carter, came to me 12th August, 1894. "When he awoke on Sunday, July 29th, he found pronounced, firm, brawny, painless swelling of one eyebrow. It disappeared gradually toward evening. The following Sunday morning, August 5th, he found similar swelling of lower part of forehead and of both eyebrows, and between 4 and 5 p.m. swelling of lower lip, not to the same extent, but noticeable. The swelling of the lip had disappeared when he woke on Monday morning; that of the brow disappeared during the day while he was at work. To-day, Sunday, 12th August, patient comes with similar swelling of upper lip, which began after dinner at 2 p.m., and reached its present height (7 p.m.) in two hours. The swelling is like that caused by a wasp-sting, being brawny, uniform, not pitting on pressure, but painless. The lip projects quite an inch below the lower, in fact as far out as to be on a level with the tip of his nose. Teeth and gums are sound. Patient never touches shell-fish; he took fish last Sunday, but not till after the swelling had appeared. He smokes three ounces of black tobacco per week; but he eats much more nitrogenous food on Sunday than on other days. He has much less exertion and more food on Sundays; and probably the same applies to Saturday evenings. He came to Hamilton only nine weeks ago: before that he used to work Sundays and week-days as a shepherd,

¹ It is of special interest to note in this connection of urticaria with asthma, that Ehrlich and Lazarus find *eosinophile leucocytosis* only in three groups of cases—(a) asthma, (b) a few skin diseases, urticaria being one of the chief, (c) the subjects of intestinal worms.

and never had such swelling. (Remember, in this connection, Bouchard's statement as to the diminished toxicity of urine after exercise in the country.) He is a strong, well-built lad, tongue clean, pulse 72 and soft, heart sound. Urine, straw colour, no albumen, no sugar." Some weeks later I saw him, and found that a similar swelling had occurred the following Sunday and slowly disappeared. He took a purgative, as advised, and never had any recurrence.

What feasible explanation is there for this case with its "week-end" periodicity other than that it was of toxic nature? And if so, then so also of "week-end" asthma. One other peculiar case of toxic origin, that of "bronchial flux," should be remembered here, but will be more appropriately considered later.

All of these instances point to a toxin of "convulsive" nature, a spasm-producing toxin, acting on the blood-vessels, and it is at least equally feasible to explain the "bilious" vomiting and the diarrhoea occurring in many cases of asthma (see p. 189) as due to a spasm-producing toxin acting on stomach and intestine, as it is to ascribe them to local irritation, though that may be and often is present.

Let us now sum up the case for the toxic pathogeny of asthma. Many cases show periodicity. That it is apt to occur at night or on waking is well known; I have shown that many cases, especially among working men, occur chiefly on Sundays or Mondays. Bouchard has shown that the toxins of urine of night periods or of periods of rest are more convulsive (spasm-producing) than the urine of day periods or of periods of muscular activity. This condition of the urine is but the expression of the toxicity of the blood. This condition of blood and of urine would exactly explain the "night" and "week-end" periodicities of asthma, and is, on the other hand, what we should expect to deduce from those periodicities. And there is no other feasible explanation of those periodicities. You cannot explain them as cycles of mysterious irritation of the nervous system. Further, there are phenomena in asthmatic patients (urticaria, deep oedema, "local asphyxia") and in allied disorders, which can be most readily explained

on the toxic hypothesis. Cases of asthma which do not show marked periodicity can be explained on the same hypothesis, because they are mostly patients whose habits are less periodic.

Diathesis means biochemical tendency. The asthmatic diathesis, on the view urged here, would mean the tendency to form more than the normal quantity of certain toxins or to fail to eliminate these rapidly enough. The accumulation of these in certain patients causes asthma, because such patients have abnormal respiratory tracts, and have, in most cases, lesions in the nose, the most sensitive area of that tract.

It is easy to understand, therefore, how reflex influences passing from orifice to viscus will find in the convulsive toxins of Bouchard just the stimulus needed to produce spasm there—in the lungs. Further, the accumulation of such convulsive toxins will explain the irritable state of the nervous system on which Sydney Martin lays all the emphasis. It is even possible on this hypothesis to explain those difficult cases of “cat” and “hay” asthma. Smells or dust will give to unusually sensitive noses the stimulus needed by such a poisoned nervous system to evoke an asthmatic spasm. Goodhart,¹ referring to the connection between *urticaria* and asthma, says, “It is sometimes associated with or replaces asthma, as a case of asthma produced by contact with cats will show. I have three records of such cases.” This connection with *urticaria* will be referred to later on.

The relation of uric acid to “convulsive” toxins remains to be considered. Bouchard² holds, and quotes experiments to show, that uric acid, even when injected in fairly large quantity, has very feeble toxic power. On the other hand, he³ shows that urea is diuretic. And there is little doubt that uric acid is not so. An interesting point in this respect can be gathered from the tables I have already given. If the average quantity of urine of asthma-free patients be compared with that of asthmatic patients, we find the latter pass much

¹ Goodhart in *Allbutt's Med.*, v., p. 291.

² Bouchard, *loc. cit.*, p. 51.

³ Bouchard, *cap. vi.*

less urine, even *including that passed in asthma-free periods*. (Only 12-hour periods have been reckoned.)

URINE, . . .	{	Asthma-free patients,	705 c.c.	520 c.c.	1225 c.c.
		Asthmatic patients,	470 c.c.	356 c.c.	826 c.c.
			Day.	Night.	Total.
URIC ACID (calculated in same way),	{	Asthma-free patients,	·215 gm.	·115 gm.	·330 gm.
		Asthmatic patients,	·311 gm.	·225 gm.	·536 gm.

And this also *includes asthma-free periods*. If the figures for asthmatic patients had been limited to asthma periods, the excess of uric acid would have been greater. Therefore, one probable influence of uric acid is that it interferes with diuresis and consequently with the elimination of "convulsive" toxins. There is also, probably, a prior relationship. Haig¹ holds that the uric acid excreted comes solely from the proteids of the food. But this view is hardly tenable in the face of recent researches.² There is every reason to suppose that it is a product of nitrogenous metabolism. If so, it is a gross one, and may be regarded as only a less complete oxidation than urea. Excess of uric acid probably means an error of nitrogenous metabolism, in which more subtle, less oxidised, and more toxic products are evolved. So that uric acid, while holding a more or less constant relationship to asthma, is probably rather an index of other toxic products, formed and accumulated in the body, as well as a hindrance to that elimination which would be better promoted by the more perfect oxidation-product and diuretic, urea. Some of these may be abnormal; but among them is probably the "convulsive" toxin of Bouchard in excess.

As to the actual condition of the lung during asthma in the light of the view here urged, it is to be remembered there are two leading theories—(1) spasm of the bronchial involuntary muscles causing contraction of the lumen of the softer bronchial tubes; (2) some rapid tumefaction of the bronchial mucous membrane. Martin, and most men, adopt the former view; Gee, the latter. Gee says asthma is "pneumonic" in nature,

¹ Haig, *Uric Acid*.

² See a *résumé* of the question of the origin of uric acid by Dr. Carstairs Douglas, *Edin. Med. Journ.*, Jan. 1900.

that the obstruction is not due to any sort of spasm, but, in Willis's phrase, to an afflux of humours upon the air passages. "Five or six affections of the respiratory tract which are closely allied to asthma" (a peculiar form of bronchitis in children, spasmodic croup, paroxysmal coryza, paroxysmal bronchial flux, hay fever), "may be said to constitute, together with asthma, a special class of diseases, characterised as follows: They set in suddenly, they speedily reach their highest point of severity, their duration is short, they are apt to recur, and in the intervals the patient is more or less free from signs of disease until the catarrh becomes chronic. All these diseases are chronic. Now, catarrh implies two conditions—increased secretion and swelling of the membrane, but neither of these conditions is constant. The defluxion or increased secretion may be absent, or at most may be very small, and this constitutes Laennec's dry catarrh. The swelling is more constant, at least in acute catarrh: in paroxysmal coryza, which has so close an affinity with asthma, the swelling can be seen: moreover both defluxion and swelling occur with great rapidity. The two concomitants of catarrh will explain the most important symptoms of all the disorders in question, including asthma."

Gee scouts the idea of spasm, but it is plain that he cannot get rid of words suggesting spasm from his terminology. The presence of more or less catarrh in many cases is undoubted; but such swelling and catarrh as he holds out for are quite consistent with Sir Andrew Clark's¹ view of a "vaso-motor neurosis" producing changes in the bronchial mucous membrane similar to those of urticaria in the skin. Martin thinks if this were so, "a profuse expectoration of a mucoid and watery liquid would occur after the paroxysm." The answer to this objection is that one would not expect it in some cases and that it does occur in others. There are great varieties in urticaria. Fitzgerald² has pointed out that in angio-neuroses affecting the skin, the swelling may be subcutaneous, cutaneous, or subcuticular, only in the last form becoming vesicles or bullae. In ordinary urticaria there is swelling, but neither pitting nor vesicles, and the centre of a wheal is usually blanched. All

¹Clark, *Amer. Journ. of Med. Science*, 1886, vol. xci.

²Fitzgerald, *Edinb. Hosp. Reports*, vol. i., p. 179.

we should expect then in internal urticaria involving the bronchial tubes is, especially at first, very little, tough secretion. And this is what happens often in asthma. Again, many cases of asthma are accompanied by or alternate with urticaria (Cases 6, 10, 14). Swelling similar to Quincke's deep oedema frequently attacks the pharynx. Fitzgerald mentions a case of acute circumscribed oedema with a history of asthma. Several interesting cases showing the connection between urticaria and asthma will be found in Ramsay-Smith's¹ book on *Angio-neurosis*. Case 6 of my list once showed an interesting angio-neurosis. There was, while patient was suffering from slight asthma, an attack similar to Raynaud's disease affecting one thenar eminence—a local asphyxia, or "asthma" of the part. The part was pale and painful, and later became dusky. The affection did not last more than 48 hours. In Case 14, which is one of sneezing, urticaria, and asthma, there was once an erythematous flush across the nose and cheeks like erysipelas, but without pain or marked swelling. It passed off in a few hours.

On the other hand, Ramsay-Smith² mentions cases of angio-neuroses of the skin accompanied by vesicles and bullae, *i.e.* free transudation of fluid, and in some cases of asthma the secretion is profuse. In Cases 7, 9, 21 it was so, and in two of them the mucus was at first viscid and ultimately mixed with blood. In the first two of those cases, *petechiae formed on the skin*. Gee,³ under the heading of "Paroxysmal Bronchial Flux," quotes a remarkable case mentioned by Beddoes, where a woman was on four different occasions seized by attacks like spasmodic asthma, but much more violent, and on their remission attended by very copious discharge from the bronchi. On the fourth occasion the dyspnoea developed very suddenly, and she appeared within five minutes almost suffocated and became unconscious for two hours, during which time a very large quantity of frothy "serum" (mucus) tinged a little with blood was discharged without any visible effort by the mouth and nostrils. Then she began to cough again, and altogether put up 3 or 4 pints of fluid. At the end of three hours she

¹Ramsay-Smith, *Angio-neurosis*, pp. 31, 61.

²*Id.*, pp. 9, 19.

³Gee, *loc. cit.*

became conscious and slowly rallied. The following seems a similar case, else I should not have thought of quoting it, as there is little resemblance to asthma.¹ It is the more interesting as being probably of toxic origin. A stoutly built lad, who drove a butcher's cart round outlying districts and who had no food but an apple between breakfast and supper, went to bed on the evening of 25th August, 1896. He looked a little pale. Next morning he was found unconscious in bed, breathing loudly, with white foam coming from mouth and nostrils. His chest was full of bubbling rales, which could be heard in the next room. There was no dulness about the lungs, the heart could not be heard for rales, but was not enlarged. He could not be roused. The pupils, when the eyelids were lifted, were found to be equal and somewhat contracted, there was no squint; the eyeballs were rolled slowly from side to side. There was no paralysis, as patient occasionally moved his limbs. He vomited a little clear mucus while I was there, and his pillow looked as if this might have happened during the night. His bowels moved involuntarily at the same time as he vomited. Six ounces clear urine withdrawn by catheter gave sp. gr. 1017, albumen, and a reaction to Fehling's solution (? sugar). He remained in the same unconscious condition all day. The rales gradually diminished, and by midnight were nearly gone, but the respirations remained very fast, 80 *per minute*, the pulse rate being about the same. The temperature in the morning was 99 (pulse 120), at noon temperature was 101·5, at 4 p.m. 99, at 6 p.m. 101·5, at 10 p.m. 99. The teeth were rigidly clenched till the evening, when he yawned, and the tongue was seen to be foul. A pint of urine withdrawn in the evening gave the same reaction for albumen and (?) sugar as in the morning. Patient was seen by four doctors, who all agreed he would die. So his mother, when she arrived from the country, burned his abdomen with hot applications, and next day, 27th, the patient slowly rallied, and towards evening tried to mutter a response to questions. Faeces were passed twice and urine once in bed. The urine got by catheter contained no sugar or albumen. Still slight cooing rale in chest. On the third day he was conscious, but somnolent; respirations 35,

¹ Yet Case 21 shows kindred points.

pulse 72. Throughout the illness he was of good colour, neither cyanosed nor pale. The burn of his abdomen took three weeks to heal. The diagnosis was at first between a brain lesion, probably involving the medulla, and a poison. The complete recovery, the condition of urine and complete absence of paralysis, favoured the idea of a toxin affecting the medulla. Patient shortly afterwards left the district, so I do not know whether he has had other attacks like Beddoes' patient or ever developed asthma.

If such cases are of asthmatic kindred, then asthma is as varied a disease as angio-neuroses of the skin. The amount of the exuded fluid varies in both. Both affections come and go quickly. Lastly, the mucous membrane of the trachea and right bronchus has been seen during asthma to be deeply injected. Observations on this point ought to be more numerous than they are, for in severe asthma the vocal cords are abducted to their fullest extent, and held so, as I have shown in Cases 16 and 20. Unfortunately, I have no note of the mucous membrane, but my impression is that it was congested. Case 9 once came feeling as if he were going to have asthma. The interior of the larynx was uniformly injected, the cords pink; and on another occasion during an attack the same condition was seen, with sticky mucus between the cords and injection of the mucous membrane of the trachea. In another case of moderate severity, where the cords were not abducted to their widest and were slightly moving with respiration, the mucous membrane of the larynx, especially about the glottis, was injected, the upper part of trachea slightly but not much injected; interior of nose very red, inflamed, and discharging muco-pus. There is much, then, to support the view that asthma may be, in many cases at least, a kind of urticaria due to vaso-motor spasm. To the other view advanced by Gairdner¹ fifty years ago, that of spasm of the "scavenger" muscles of the finer bronchi, there is no real objection. There is no evidence against it; it satisfies the clinical facts; the muscles referred to are found in the autopsies of such cases to be hypertrophied. (It is to be noted, however, that thickening and hyperaemia of

¹ Gairdner, *On the Pathology. Anat. of Bronchitis, etc.*, part ii., pp. 39 seq.

the mucous membrane are also found.) The theory of bronchial spasm, in short, is supported by anatomical, pathological, and experimental evidence; to reject it, as Gee does, because such spasm has not been seen, is absurd, and an asking for evidence that can never be got. Likewise the theory of vasomotor spasm cannot be rejected, for such spasm is frequent in asthmatic patients, and it also accounts for many facts of many cases.

There is no need to choose between the two theories. If asthma is due to a "convulsive" toxin, the difficulty would be to understand why such toxin should not act both on bronchial muscles and on those of the blood-vessels, possibly more at different times and in different cases on one than on the other. Nothing better can be urged now for this view than what was urged fifty years ago by Gairdner for his own theory. " 'The contractility of the bronchi,' says Dr. Williams, 'resembles that of the intestines or of the arteries more than that of voluntary muscles or of the oesophagus, the contractions and relaxations being gradual and not sudden.' This kind of contractility is precisely that which empties the arteries of their blood after death." And since, as I have shown, there is in some cases spasm of stomach and intestine, causing vomiting and diarrhoea, of smaller blood-vessels causing petechiae, local asphyxia, urticaria, deep oedema, why then may there not be spasm of the bronchi and of the smaller blood-vessels connected with them, producing sometimes merely dyspnoea, with little secretion, sometimes dyspnoea with much, and occasionally blood-stained, secretion?

The conclusions then to which I would come are:

1. That in asthma there are two factors:

(a) A lesion in the respiratory tract.

(b) A toxic condition of the blood.

2. That unless both are present there is no asthma. Sometimes the one factor, sometimes the other, is the more important.

3. That there is in most cases an abnormal condition of the nose, which is important, because the nose is the respiratory orifice and the most sensitive part of the respiratory tract.

4. That the "convulsive" toxin normally present in the urine and therefore present in the blood, is probably in excess in cases of asthma. There may be others in addition to potash.

5. That excess of uric acid has a direct connection with asthma, and may have some direct effect in producing spasm; but more probably represents imperfect metabolism and excess of the normal "convulsive" toxin or the presence of others, and more probably acts by hindering elimination of these.

6. That in many cases of asthma spasm of the smaller blood vessels does exist in other parts than the lungs; and, in some cases at least, probably exists there as well, together with spasm of the bronchi, which probably is present in all cases.

The treatment of asthma naturally divides itself into two parts—immediate treatment of the paroxysm, ultimate treatment of the disease. In both cases we have to consider the double factor—the lesion in the respiratory tract, the poisoned condition of the blood.

1. *The paroxysm.*—If possible examine the nose. If the mucous membrane is injected, and especially if the two inferior turbinals are turgid, apply cocain solution 5 per cent. to 15 per cent. on swobs, using the weakest solution which will give an anaesthetic effect. In asthma usually the stronger solutions are necessary. Swobs are safer than the spray, though less easy to apply. I usually combine 5 per cent. resorcin with the cocain: this keeps the solution fresh and increases the anaesthetic effect. Then apply suprarenal solution. This does not keep well, and is best made fresh every day or two. Grind a gr. 5 tabloid of suprarenal extract in a drachm of boiled water: the solution is ready in a few minutes, though it is best to have it ready beforehand. To do good it must be thoroughly applied on swobs, and the interior of the nose should be seen while applying it. Cocain without suprarenal, or *vice versa*, gives a much diminished effect. They cannot be used in combination, because they decompose each other. Solis Cohen for his own asthma dissolves a suprarenal tabloid under the tongue every 4 or 6 hours, and speaks highly of the

result. I have tried it in this way with less effect than by the nose. (Given by the stomach, suprarenal is apt to sicken in much the same way as zinc sulphate.) Treating larynx and base of tongue in similar fashion sometimes eases the spasm. Suprarenal solution is said to be specially useful in hay asthma, and this would be an advantage, for cocain is a snare and often necessitates the use of stronger and stronger solutions. Where nasal treatment fails, inhalation may be tried. Amyl nitrite sometimes eases, often fails, sometimes aggravates. Many patients, after trying all the doctor tries, fall back on asthma powders composed of stramonium, lobelia, nitre, etc. The cigarette is the best form in which to use these. The effect of these powders is said to depend on the formation of the nitrous fumes and of pyridine. An American doctor, evidently acting on this idea, extols the following prescription, which he used as spray for his own asthma, but which I have only once found successful :

Antipyrin,	gr. v.
Pyridin,	℥20.
Sod. Nitrit.,	gr. 40.
Tr. Lobel.,	Tr. Stramon.,	Tr. Bellad.,	Vin. Ipec.,	āā,	℥100.			
Glycerin,	℥ss.
Aq.,	ad ℥iss.

Paraldehyde internally is often successful. When there is much functional secretion a smart effect is often got from mustard-bran poultices to the chest. But poultices are worse than useless in most cases of asthma.

Treatment of the condition of the blood.—A saline purge is often needed. For internal medication the best drug is pot. iod. or sod. iod. in combination with others. Except when there is much uric acid being excreted, I always combine it with liq. arsen., and often add tr. hyoseyam., and, if there is not much bronchial secretion, vin. ipec. Instead of the last two drugs pot. brom. may be substituted with advantage. When there is marked uric-acid excess sod. salicyl. is sometimes the best combination with sod. iod. I have known this give more relief than the ordinary mist. iod. et ars. ; but many people cannot take it. In view of the fact that potassium is a

"convulsive" poison, it may be an advantage to use combinations with sodium in treating conditions of spasm like asthma.

2. *Treatment of the disease.* *Local.*—Semon¹ has recently given a very hesitating view as to the value of intra-nasal treatment in asthma. He says, "the *proportion* of really successful cases in my experience is *very* small compared with those which are only temporarily benefited, and even much more so in proportion to the absolutely unsuccessful ones." (This, however, is only another way of saying that there is always more than one factor present in asthma, and that often the more important and the more difficult to treat.) He therefore explains to patients "the present state of the whole matter without either urging or dissuading from intra-nasal treatment." How I prefer to put it is: Asthma depends on more than one factor—simply treating one rarely cures; but there is little hope of curing when the nasal condition is obviously unsatisfactory, unless that be treated; the more pronounced the nasal condition the more hope of real, but it may not be lasting, benefit. Two things must be remembered before condemning intra-nasal treatment: (1) the difficulty of being thorough without doing too much; (2) the strong tendency of nasal lesions to recur.

What you wish to procure in intra-nasal treatment is that turbinal structures shall not touch septal structures, and to prevent engorgement. There is the risk, however, if too much turbinal be removed, of creating a condition similar to that in atrophic rhinitis, a condition worse than the first. Especially is this the case if the cautery be freely used, against which there has lately been much outcry, and yet it is most useful if recurrence is to be prevented. The difficulty of preventing recurrence of intra-nasal disease is greater than would be readily imagined; hence intra-nasal treatment is often brought into discredit, whereas the right thing is to have it repeated. (See Case 3, where, by the removal of polypi, the asthma was practically cured until the polypi returned.) Lastly, as stomach and intestinal disorders can often be best prevented by beginning at the mouth, which is too often neglected, so

¹ Semon, *loc. cit.*

nasal disorders should not be allowed to stand as if of no consequence, and in this way bronchial troubles and asthma may be prevented.

Two details in intra-nasal treatment are worth comment. (1) The use of suprarenal solution shrinks and blanches engorged structures, leaving inflamed tissue red and prominent. It also, by preventing bleeding, gives a good view of the parts and facilitates a longer operation. Its disadvantage is that you do not know how much bleeding will occur till two hours after application. This has been miscalled a reaction. (2) Asthmatic patients commonly have very irritable noses; hence plugging should be avoided where possible. Plugging is essential after removal of inferior turbinal tissue, but not in the case of the middle turbinal, unless the operation has been extensive or there are other indications. The insufflation of aristol and a dossil of antiseptic wool in the nostril are usually sufficient after middle turbinal operations. The latter the patient can replace at will, and sniff up more aristol. (Patients 12 and 16, who had previously been treated by specialists, expressed their delight at the comfort they got from the use of the suprarenal and the absence of the plug.)

General treatment.—As asthma, with its slowly poisoned nervous system, is not made in a day, treatment must usually be prolonged. The important thing is to try to prevent toxic conditions of the blood, and so anticipate paroxysms of asthma. Where asthma is of the "week-end" type, a mercurial on Saturday or Sunday night, followed by a saline next morning, often prevents an attack. Blue pills seem better than calomel, which sometimes aggravates. Case 7 says that whenever she notices her motions getting darker she knows to take blue pill, and to this as much as to intra-nasal treatment she ascribes her cure. Apart from this, the medicinal treatment is much the same as that given under treatment of the paroxysm. Arsenic and iodide form the best routine combination; salicylate and iodide, if tolerated, when there is marked uric-acid excess. The dietetic treatment has been worked out by Hyde Salter. It is often that for uric-acid excess. Haig would eliminate red meat entirely, but it is to

be remembered that this is the item in which most iron is conveyed. After what has been said on "week-end" asthma, a sufficiency of open-air exercise is obviously important, as well as a cutting down of the diet at resting times, both in the evening and at week-end. Cod-liver oil often benefits, especially adolescents. In some of the appended cases it seems to me that the cure has resulted from a steady improvement in the whole general condition as the patient has grown to maturity.

Climate has much to do with asthma. This district has a moist, relaxing atmosphere, dense clay soil, and lies in a hollow. Hence the frequency of nasal disease and so of asthma. If there is one thing which will produce a sodden condition of the nasal mucous membrane it is moisture. For this reason lotions should rarely be used for the nose. It is difficult to select a climate for each individual case of asthma. Much will depend on the chief factor in play in the disease. When there is oedematous hypertrophy of the nasal mucous membrane a dry climate is obviously best. Where pulmonary emphysema is great, the place selected should also be at a low altitude, though Case 6 and another of emphysema without asthma have greatly benefited by going to Denver, which is rather high for emphysema.

To sum up, most cases can be benefited (1) by suitable nasal treatment, and (2) by treatment directed to prevent the undue formation of toxins and uric acid and to secure their prompt elimination. As a rule, the most difficult cases to aid are those of long-standing bronchitic asthma without marked nasal disease, or those with an inveterate tendency to uric-acid excess yet without marked nasal disease. Finally, it is not meant that every nasal abnormality needs treatment. (*E.g.* spurs can often be left alone.) A good but not invariable guide is to be got from the effect of cocain and suprarenal solutions in allaying the asthmatic paroxysm.

Postscript.—The above was written in May. I wish to add some remarks as to treatment which I have since found useful. In certain *selected* cases I have proved the value of Dr. Wm. Murray's treatment of asthma. Case 22 has

never had asthma since this was begun; any benefit I had hitherto been able to give was trivial and fleeting.

R.	Tr. Stramon.,	5ii.
	Sod. Carb.,	5iii.
	Ammon. Carb.,	Mag. Carb.,	.	.	.	āā 5i.
	P. Rhei.,	gr. xx.
	Chloroform,	℥xx.
	Aq. M. Pip.,	ad 5viii.

3 ss. t.i.d. for 4 to 6 days, and then for 3 months once at bedtime, with ℥ v. Liq. Arsen. after breakfast and dinner.

(See that valuable book, *Rough Notes on Remedies*.)

Lately a proprietary "cure" for asthma has had a great vogue. In allaying the paroxysm the remedy is wonderfully successful. Its success depends on the fact that it (1) is a potent remedy (2) directly applied to the respiratory tract (3) by a proper apparatus. Its chief ingredients are cocain and sodium nitrite. It is very properly applied through the nose in a very fine spray. Most instruments on the market are not safe to use with so powerful a remedy. The spray should be the finest possible cloud, so as to be readily carried on the respiratory current, should not be of greater speed than that current, nor of too great force, else it will coat in excess the upper air passages and fail to reach the lower. The apparatus referred to, as also Oppenheimer's, fulfil these conditions.

CASES.

With regard to these, three points call for note:

1. *Diagnosis*.—This could fairly be disputed, I think, only in Case 19. I should hesitate to call it asthma; it seemed more like nasal obstruction, with symptoms similar to those caused by adenoids. Yet if this were so, it is difficult to understand the constant recurrence of distressed breathing at night. I have seen worse nasal obstruction without such distress. However, this case has been omitted in calculating percentages.

2. *Nasal treatment* was sometimes too protracted for various reasons, but chiefly to avoid keeping patients off work.

3. *Results*.—"Cure" means no return of asthma. "Practical cure" includes (a) cases where patient had no recurrence of real paroxysmal asthma, but felt "tightness in breathing," as if he would have had asthma had he not resorted to medicinal treatment; and (b) cases formerly subject to severe asthma, and remaining free of it unless on taking a severe cold (Cases 7, 12); *e.g.* Case 7 had been off work seven years owing to asthma; treatment has enabled her to keep at work for the last three.

Including Case 19, we thus get:

- 7 cases of "cure."
- 4 cases of "practical cure."
- 9 cases of improvement sometimes great.
- 4 cases of no improvement.
- 1 case still under operative treatment.

(1) Of the seven cases of cure, the treatment was purely nasal in four. Case 1 was not seen for long enough afterwards to make me feel thoroughly satisfied with it, but the patient was satisfied, and rather resented my wishing to see him further. He had no more asthma while I knew him. Case 13 was one of rather pronounced asthma. He has had no asthma for a year. In Case 18 the asthma was slight. Case 19 may not have been asthma. (See above under "Diagnosis.") In these four, treatment was purely intra-nasal. The remaining three cases were all adolescents. In two of them (5 and 8), treatment was chiefly nasal, but some attention was directed to the general health and diet. In Case 9 much credit is to be given to his having become able to take maltine and cod-liver oil, the first preparation of the oil he has ever been able to take: this credit is shared equally by the nasal treatment, and, I think, by his going to sea as an engineer. (2) Four cases of practical cure.—In Case 3 the treatment has been chiefly nasal. After the polypi were removed she had no real asthma, but often at bedtime took a dose of her iodide mixture to prevent an attack which she thought impending. When polypi recurred, so did asthma. It has again improved since they were treated. In Case 16 asthma was severe till the polypi were all removed and the nose healed. In the last six or eight months there has been only one attack of asthma, while

patient had a bad cold. Nothing but nasal treatment did this patient any real good. Case 7 was one of severe asthma, which has been practically cured by nasal treatment, and by blue pill and salines at more or less regular intervals. The general health was watched. Case 25 ascribed her cure entirely to mist. iod. et ars. (3) Nine cases of improvement.—In all, the improvement has been distinct, sometimes great, enabling patient to work regularly. Those that have improved most are those who have had marked nasal disease. In Case 6 the improvement has resulted from a change to Denver, and from that alone. In Case 23 no treatment did any real good, but there has been no asthma since an attack of pneumonia two years ago. Of these two cases, the former had not pronounced nasal disease, the latter none. Two cases (4 and 16) are still under treatment, but have been long enough under observation to justify their being classed here. (4) Four cases of no improvement.—22 is a case of malnutrition without marked nasal disease. Asthma of two years standing has been little benefited except by treatment directed to digestion, and is not likely to improve while patient is in her present circumstances and low-lying house. 10 is also a case of malnutrition (less decided) and of long continued uric-acid excess. Nasal treatment declined. 14 is a case whose health has been steadily drained by a bleeding fibroid. The nasal tissues have long been very irritable and somewhat hypertrophied. This is a case of no improvement. But thorough treatment has not been accepted. Asthma slight. 25 is a boy with “hereditary” asthma. Bronchitis has been frequent: some intra-nasal hypertrophy. Treatment declined. (5) One case (21) is classed as still under treatment, because the nasal treatment was started not long ago, and attacks of asthma have not been frequent.

Postscript.—Remarks have been added where necessary to alter the narrative up to date (from May to December).

CASE I.

Male, aged 30, miner. Severe asthma, fibro-mucous polypus, removal of which cured asthma. This case has been sufficiently

mentioned at pp. 154 and 155. I regret I have no subsequent information.

CASE II.

Male, aged 22, shoemaker, Hamilton. 1st May, 1899. An under-fed, ill-grown lad, with stooping shoulders, and bronchitis from childhood, developed regular paroxysmal asthma when he went to work at the age of 14. Attacks, accompanied by "bilious" vomiting, occurred nearly always on Mondays, preventing work on that day. (Much more rest and food on Sundays than on week days, when he had to walk four miles to work.) Was first seen June, 1893, when the hypertrophied nasal mucous membrane was treated, but not thoroughly. Asthma became less frequent, but not less severe. In 1897 he started business for himself, had meals more regularly, and on Sundays more exercise. The Monday periodicity, which had been such a striking feature, then disappeared, so that he would be off work only once in a few months, and then for several days; but there are few nights on which he has not slight asthma. He has usually to burn "asthma powder" on going to bed, perhaps once during the night, and again on waking, when he is apt to have a more pronounced attack. But for the inhalation he does not think he would sleep. There are the usual signs of marked emphysema, the cardiac dulness being almost obliterated. The nostrils, except a little in the right, are blocked by hypertrophied oedematous mucous membrane, especially over turbinals, and by a large spur in left nostril. Constant dripping of watery mucus. Frontal headache and "creeping" feelings in scalp point to extension of disease to sinuses. The nasal membrane is beyond hope of restoration to normal, that of pharynx is thickened, that of larynx and trachea slightly injected. Patient has become addicted to drink.

1st August, 1899. On 15th May septal spur was removed, and during the following 4 days he had several attacks of asthma relieved by applying cocain and suprarenal solutions inside nose. Within 15 minutes the ease to respiration, especially to inspiration, and the cessation of cooing rales, was striking. He went out one night, caught cold, and took pneumonia

(20th May). During that illness and the succeeding convalescence at coast he had no asthma, but since the second night after his return (8th July) he has had slight attacks at bedtime.

26th January, 1900. On different occasions hypertrophied nasal mucous membrane has been removed and cauterised. While there has usually been asthma the day after an operation, patient has been more free of it since removal of septal spur 8 months ago than for years. Suprarenal and cocain solutions have given striking relief when required.

18th April, 1900. Probably owing to his drinking habits patient did not return for treatment, and early this month again took pneumonia, of which he died to-day. Asthma rather troublesome during February and March, and much remained to be done for nose.

CASE III.

Female, 66, Wishaw. 18th January, 1900. Bronchitis, frequent and severe, began 30 years ago; asthma 13 years ago. Nasal polypi first known to exist 16 years ago. Asthma, slight at first, gradually became so severe that her friends have, once at least, been summoned to her deathbed. Cold in head always ends in bronchitis and asthma. Her doctor, though aware of the polypi, did not seem to think them an important factor in the case. Patient first came to me 6 years ago, when I removed polypi from both nostrils. For the next 2 years she had no asthma even when she had bronchitis. There was never more than "tightness of breathing," probably because she always kept *mist. iod. et. ars.* at hand. During last 3 years there has been slight asthma, and patient returned last November with polypi closing both olfactory clefts. These I removed, as also a long ridge in left nostril opposite left inferior turbinal which was causing much irritation and obstruction. Cauterised base of polypi. In right nostril is a prominent septal "spine," but far back, and causing no irritation.

18th May, 1900. There was a slight attack of asthma during healing of septal wound, but even though she had a severe, prolonged bronchitis, and though, in spite of the cautery,

polypi are again budding, there has been "immense" improvement in the asthma, and no bad attack since last note. Polypi removed and cautery applied.

CASE IV.

Male, aged 35, miner, Hamilton. 17th September, 1899. Has had asthma for 5 years, especially when he gets out of sorts during an east wind or has disordered stomach. The digestive disorder and the asthma are closely connected, for asthma is very frequent in the mornings just after he gets up, and any food taken in the morning is vomited, so that he usually goes to work without breaking his fast, and carries some food with him. Asthma is specially bad on Tuesday mornings: he has more food and at other hours on Sundays than on week days. Nose: a septal twist leaves right nostril fairly free except where it impinges on middle turbinal, but, together with a spur, quite occludes left nostril. This spur is really a long ridge with a sharp spine at one part indenting inferior turbinal. Turbinals inflamed. Uric-acid excess.

26th May, 1900. At different times the septal ridge and spur were removed, and turbinectomy of left inferior and middle turbinals performed, leaving a useful nostril. During this treatment asthma gradually became less frequent and severe, so that by the beginning of February he was, for the first time in 6 months, able to do a full week's work. As he became regular at his work asthma recurred regularly on Mondays for 5 weeks, then disappeared for a month. He has had occasional attacks lately, but does not lose more than a day's work. These, during the period of intra-nasal treatment, have been somewhat relieved by applying cocain and suprarenal solutions to the left nostril. Digestive disorder, with marked flatulence, has been rather persistent, and treated with only partial success. The general health has improved; this may partly account for the improvement in the asthma.

P.S.—December, 1900. There has been recurrence of asthma, but not to the same extent as formerly, and greatly relieved by spray of pyridin, etc. Still the same tendency to vomit in mornings.

CASE V.

Female, 21, Bothwell. 18th May, 1900. When first seen in 1895 patient was anaemic and complained of nasal obstruction, frequent sneezing, and asthma. Bronchitis began in infancy, asthma at the age of 3. Polypi and hypertrophied nasal tissue were removed, and cautery and chromic acid applied; cod liver oil and iron given with distinct improvement in general health. The nasal obstruction was relieved; asthma disappeared. Lately there has been indigestion and recurrence of sneezing, and a slight feeling of nasal obstruction due to hypertrophy of both middle turbinals, and an indication that polypi may recur. Still no asthma.

CASE VI.

Male, 38, architect, Hamilton. 15th May, 1900. Bronchitis since childhood; bronchitic asthma during at least the last ten years. (Father seems to have had asthma.) Patient has often been off work lately owing to bronchitis and asthma. In September, 1897, while suffering from asthma, he had "local asphyxia" of right thenar eminence, the part being first blanched and painful, and later dusky. This did not last above 48 hours, and never recurred. In February, 1898, a severe "cold in the head" led to bronchitis; as this was passing off, asthma came on, accompanied at night by urticaria. He often complains of "coldness" in the scalp and back of neck, and of difficulty in keeping warm (cp. Case 3). Two years ago it was noted, "There is marked emphysema of the lungs, cardiac dulness is obliterated; very deficient R.M. at right base. Both inferior turbinals and right middle turbinal are hypertrophied; a thickened spongy area on each side of septum pits on pressure, and causes sneezing when touched." I advised intra-nasal treatment and change of climate, to Egypt if possible. Two specialists both agreed as to the intra-nasal treatment, which was carried out without benefit. Dr. Samson Gemmell found collapse of lung at right base, and ordered patient abroad as a matter of urgency. He went to Denver at the end of 1898, where he is said to have much improved.

P.S.—Patient has lately died of "pneumonic phthisis."

CASE VII.

Female, 34, teacher, Uddingston. July, 1897. Patient comes complaining of severe asthma. She had been troubled with bronchitis until 21; asthma began at 27, and has kept her off work for the last 7 years. Before an attack her motions became dark, her urine muddy: about 4 a.m. she will wake, feeling "tightness" in the chest, tingling of the skin, and an indefinite irritability. She will pass pale limpid urine frequently and in great quantity, and in an hour or two will have pronounced asthma. She has to sit up in bed, the slightest movement causing the greatest distress; skin and nails get livid, petechiae form, the veins stand out, there is profuse perspiration, great palpitation, and the patient sometimes swoons. Spit is scanty, tough, and sometimes blood-stained. There is much cough, and great difficulty both of inspiration and expiration, chiefly the former. There is usually "bilious" vomiting and diarrhoea. Menstrual discharge may appear. After 6 or 8 hours of the polyuria, the urine again becomes scanty and loaded with urates. Her skin during an attack is harsh and gives off a profuse, branny desquamation; her hair gets coarse. After the attack the skin becomes fine and velvety, the hair soft. An attack lasts 36 or 48 hours, and leaves her quite exhausted. She will have 4 or 5 attacks like this during 4 to 8 weeks: and then be free of asthma for a short time.

There is slight emphysema, no albuminuria. In the left nostril are mucous polypi and hypertrophy of the membrane over inferior and middle turbinals. She says the nose feels swollen and congested: she has constant sneezing.

15th May, 1900. The polypi were removed, the hypertrophy reduced. She was told to take once a week blue pill at bedtime, followed by a "Seidlitz" next morning, and during the week mist. iod. et ars. \bar{c} hyoscyam., and to inhale amylnitrite during attacks. There were several attacks at beginning of treatment, relieved not by amylnitrite, but by a few doses of the iodide mixture. She has, however, been able to ward off attacks by taking blue pill once a fortnight, and especially whenever she feels any "tightness" of breathing and notices motions becom-

ing darker or urine muddy. To this chiefly, but also to the nasal treatment, she attributes her immunity from asthma. She has had one or two mild attacks (*e.g.* after a quinsy or after a big "feed" of fruit, etc.); but whereas she was off work 7 years before treatment, she has been able to work continuously for nearly 3 years; and she once had bronchitis without asthma. There is still thickening of the anterior ends of both middle turbinals so as to impinge on septum, but the inferior remain well.

CASE VIII.

Male, 33, ironworker, Airdrie. 22nd October, 1898. Asthma began 10 years ago, for which patient first visited me 3 years ago, and I removed hypertrophied membrane from both nostrils and sent him to Dr. Walker Downie for cauterisation. This was done, and for a year patient had complete relief, but lately asthma has recurred, especially in wet weather. Sneezing has also been troublesome, especially when he washes his face or is exposed to dust. Sunday is nearly always his worst day. Meals are the same Sunday and week day, but he has no work between Saturday, 1 p.m., and Monday morning. On the rare occasions when he works on Sunday he has no asthma. There is again hypertrophy of right middle turbinal and of posterior end of right inferior turbinal; there is a thick soft patch on left side of septum. Cautery to right middle turbinal. To take 2 gr. calomel every Saturday night.

18th February, 1899. There was bad asthma for two days after the cauterisation, but since that only one slight attack on Sunday, a week ago. Sneezing gone.

12th May, 1900. Owing to distance and absence from home, patient did not have intra-nasal treatment finished, but has continued much better, though he sometimes has to use an "asthma cigarette" in the mornings for a "stuffy feeling in chest." Asthma used to last a whole day or several days. There is still hypertrophy of both middle turbinals, and the right is threatening to become polypoid again.

CASE IX.

Male, 20, engineer, Uddingston. 10th February, 1898. Patient comes on account of asthma which has troubled him since childhood. Last year he was not a fortnight free of asthma till the summer. During a bad attack his face gets livid, petechiae come out; he has headache, somnolence, bilious vomiting, and becomes very weak. The attacks are nearly always on Monday mornings. (He has an asthmatic friend 4 miles off, whose attacks often synchronise with his own.) The soft tissue of all the turbinals is hypertrophied; there are enlarged veins on both sides of the septum, which is very irritable, a touch causing sneezing even after weak cocain solutions.

May, 1900. The nasal hypertrophy was reduced by snare and cautery, especially in right nostril, where both ends of inferior turbinal had to be removed. At first calomel, later blue pill, was given on Saturday nights. Until September, 1898, there was sometimes asthma. Once, just when patient felt an attack impending, the interior of the larynx was seen to be injected; during two other attacks there was also seen sticky mucus between the cords and injection of tracheal membrane. The interior of the nose was also at those times much injected and irritable, the turbinals refusing to shrink under even 15 per cent. cocain; the tongue was furred. In September, 1898, he became able for the first time in his life, and after unsuccessful attempts on other preparations of cod-liver oil, to take the combination with maltine. His general condition began to improve, and from that time he has had no asthma.

CASE X.

Female, 37, Shotts. 12th March, 1900. Patient had laryngismus stridulus and night terrors in childhood; has had a very irritable nose since she was 19, working with wool causing intense tickling in the nostrils and sneezing. She began to have frequent colds in the head after coming to Hamilton 15 years ago, much less frequently since going to Shotts 3 years ago. Asthma began after the birth of her first

child 7 years ago. A few severe attacks have since occurred, perhaps once a year, but slight attacks (breathlessness and wheezing) are frequent in the mornings. She has a very itchy skin, and often urticaria. There is more or less constant deposit of uric acid or urates in the urine. Both right turbinals are hypertrophied, the middle impinges on the septum, the inferior is pressed against a large septal ridge. This nostril tends to drip a watery mucus whenever she stoops. The pharyngeal tissues are thickened and congested, the uvula has been amputated; under surface of epiglottis injected.

14th May, 1900. Nasal treatment declined. Mist. pot. iod. et ars. gives some, but sod. iod. and sod. salicyl. give prompt and decided relief from the asthma.

CASE XI.

Male, 20, bricklayer, Kirkmuirhill. 21st September, 1895. Asthma since childhood during the summer till 4 years ago. Since that he has had it yearly only during August and September, but escaped entirely 3 years ago. There is great respiratory, especially inspiratory, difficulty, and wheezing. In winter he never has asthma, but often "cold in the head." Digestion good, except after asthma begins. There is a polypoid condition of both middle turbinals.

2nd November, 1895. Polypi were removed, chromic acid applied. Mist. pot. iod. et ars. prescribed. No asthma for a month. The addition of tr. hyoseyam. and vin. ipec. to the mixture was a great help.

CASE XII.

Male, 49, blacksmith, Ferniegair. 29th July, 1899. Patient never had asthma till December, 1896, after which it was very persistent till June, 1898. He first became aware he had nasal polypi in September, 1897. During the next 3 months polypi were repeatedly removed at Glasgow Ear Hospital, but asthma persisted till June, 1898. It returned in February, and has been very persistent. The attacks are very bad; usually come on at midnight, waking him from

sleep. For months, even after the nasal operations, he could not sleep except sitting in a chair. He went to work at 6 a.m., often in great distress, and often having to stop on the way. On returning to breakfast at 9 a.m. the asthma would be going off. He never had asthma during the day. Monday night and Tuesday morning is the worst time, and a time he rarely misses having an attack. He takes tea four times a day; butcher meat or water seldom; potatoes, porridge, buttermilk, soup are apt to cause asthma accompanied by belching up of bitter liquid. Till asthma started he never had a cough, but had constipation. Since that bowels have been regular.

Since patient first came, last March, I have removed from both nostrils some 30 polypi and divided a synechia which had formed between left middle turbinal and septum. The whole mucous membrane, especially in right nostril, can now be seen to be thickened and diseased, and will never be restored to normal.

24th August, 1899. The anterior end of both middle turbinals have been removed and the hypertrophy otherwise treated. Probably as the result of the operations, which were on Saturdays, the tendency to asthma being worst on Monday nights and Tuesdays, has been even more marked than before treatment.

27th May, 1900. Only one slight attack of asthma, after a cold, during last 9 months. This result is due to nasal treatment, as medicinal treatment, except sulphur internally, never seemed to benefit; it is to be noted that heart and lungs seem normal; there is certainly no marked emphysema. Internal administration of suprarenal aggravated the attacks, as did draughts of water, given at bedtime on account of patient passing rather a small amount of urine (14 ozs. every 12 hours with great regularity, and with excess of uric acid). Patient advised to return at intervals in case polypi recur.

CASE XIII.

Male 54, painter, Hamilton, 14th October, 1897. Complaints of constant "cold in head," and frequent sneezing followed by asthma. Hay fever began 16 years ago; violent

sneezing and coryza every summer if he goes into the country or is even approached by a farmer in "hay time." Asthma nearly always follows, and latterly has occurred in winter as well as in summer, if he is exposed to dust or laughs heartily. Attacks are most frequent on Sundays. Lungs emphysematous, cardiac dulness obliterated, breathing mostly diaphragmatic. Polypi in nostrils, mucous membrane spongy and thickened, specially irritable at certain spongy spots on septum. Uric-acid excess on Sundays and Mondays.

15th May, 1900. Polypi were removed, their bases and the irritable septal spots cauterised. No asthma during last year, and none during the preceding two years, except once or twice after laughing heartily. Polypi recurred in 1898 and 1899 and had to be treated, and the cautery reapplied to septum. Sneezing now seldom occurs.

Note.—Patient's son, aged 30, came 28th August, 1897, complaining of paroxysmal sneezing and coryza of 6 years' duration. No asthma. Hypertrophy of nasal mucous membrane was treated by cautery and spray of camphor-menthol in parolein. Sneezing cured.

CASE XIV.

Female, 40, Hamilton. 15th May, 1900. Patient had no nasal trouble before coming to Hamilton 9 years ago. Since that her health has been kept down by a bleeding uterine fibroid. During the last 5 years she has been subject to chronic pruritus and urticaria, and to exhausting sneezing fits with profuse coryza, especially on washing her face in the mornings. There has also been nasal obstruction. Within the last year she has had, for the first time, a few slight attacks of genuine asthma. The nasal membrane is red and very irritable, especially on septum; the turbinals, especially the inferior, are much hypertrophied. Intra-nasal treatment with the cautery was advised first by myself, afterwards by Dr. Walker Downie, and was done partially, but patient shrinks from thorough treatment. There is no improvement in the nasal and general condition.

CASE XV.

Male, $5\frac{1}{2}$, Hamilton. 25th May, 1900. Bronchitis first attacked patient when a year old, and became asthmatic in type when he was $2\frac{1}{2}$. Attacks came on mostly at night, and about a year ago occurred nearly every night and were severe. Dr. Walker Downie removed adenoids and the hypertrophied anterior ends of inferior turbinals last September. Asthma ceased for a week, then recurred in milder form once every week or two. Removal of the adenoids has caused improvement in general health, which has probably helped the asthma. Patient can sleep with mouth shut, but still has some nasal obstruction. Two months ago whooping-cough began and still continues, but asthma has been absent throughout.

CASE XVI.

Male, 21, lawyer's clerk, Greenock. 15th October, 1900. Patient has had bronchitic asthma since childhood, worse at night and specially bad on Sundays; never has cold in head without asthma; has no digestive trouble. Nasal polypi were removed 5 years ago by a Glasgow specialist. A fortnight ago a doctor told him he had no polypi, but there are polypi in both nostrils, oedematous hypertrophy of all four turbinals, polypoid enlargement of the posterior end of right inferior turbinal, and a large ridge on right side of septum. Frequent uric-acid excess.

31st March, 1900. Only one attack of asthma since I began to operate, the result probably of taking a walk and catching cold the same night as an operation. Marked dyspnoea, with great respiratory difficulty; glottis wide and still; chest hyper-resonant, full of snoring rales; cardiac dullness small, liver dullness $1\frac{1}{2}$ inch in nipple line. Cocain and suprarenal solutions applied to interior of nostrils within 5 minutes caused the snoring rales to disappear, leaving only soft moist rales chiefly at right axillary base; cardiac dullness increased, the hepatic to 3 inch; glottis less dilated and slightly mobile; a free airway opened in both nostrils. Apart from this attack he has had no

"asthma," but often a slight "tightness" on waking on Monday mornings.

26th May, 1900. Has had a third attack of asthma since treatment began 7 months ago, probably the result of an operation, the second being set up by a violent dust-storm. That is, he has been better since treatment began than in his life before. Polypi, hypertrophied parts of the turbinals, and the septal ridge have been removed, and cautery used. Mist. iod. et ars. with pot. brom. and tr. hyoscyam. given internally; also cod liver oil. Turbinectomy of right middle turbinal ought still to be done.

P.S.—October, 1900. Patient remains very well.

CASE XVII.

Male, 17, Bothwell. Came last October on account of asthma of 9 years' standing. An attack begins with cold in head and incessant coughing, and ends in bronchitis. He over-eats. Some years ago he was put on skim milk; attacks became less severe and less frequent, lasting only two or three days and often not ending in bronchitis. When 10 he spent two months at Musselburgh and had asthma the whole time, but no bronchitis. The same occurred for 5 months he was at Mentone. Sneezing was not nearly so common as incessant coughing at the beginning of an attack. Patient was a mouth-breather, the nasal obstruction being due to a twisted septum. This was removed by Dr. Downie last October, and asthma has not recurred.

P.S.—December, 1900. An attack occurred last August, the result of over-feeding during holiday. While at school he has much more outdoor exercise and more restricted diet.

CASE XVIII.

Female, 50, Glasgow. 14th November, 1898. Has had "a stuffy feeling" in nose for many years; takes "cold" in the nose readily; has a tendency to asthma in the evenings. Both middle turbinals are hypertrophied; polypus and spur in left.

May, 1900. Reports herself very well; no return of asthmatic symptoms since nasal treatment about time of last note.

CASE XIX.

Female, 39, Ferniegair. 15th May, 1900. This case has been referred to under "Diagnosis." It is probably rather one of dyspnoea resulting from nasal obstruction, similar to that of adenoids, than asthma. It is worth inserting for that and other reasons. Patient came in June, 1898, complaining of difficulty of breathing through the nose of 2 years' standing. The dyspnoea was always worse at night; she would start up from sleep gasping for breath, and for 7 months had not had one complete night's rest. While swallowing she felt as if she would choke. She was seen by several medical men, who did not seem to understand her distress, and at Glasgow Western Infirmary she says she was told she was suffering from "depression of spirits." From the right nostril I removed a single large polypus and a smaller from the left. The night after the larger was removed she slept as she had not done for years, and there has been no dyspnoea since. She never called her trouble "asthma." Within the last 17 months she has had attacks of peripheral neuritis, first in right hand and forearm, and later and repeatedly in left. The symptoms were numbness, tingling, feebleness, intense pain, a feeling as if the hand would burst, tenderness over the nerves, slight anaesthesia and analgesia, increased reaction to galvanism: they are suggestive of erythromelalgia, and come on at night after a day at the wash-tub. The condition is of interest in relation to the idea of asthma and to the attack of Raynaud's disease in Case 6.

CASE XX.

Male, 39, pipe-layer, Hamilton. 2nd September, 1899. Eight years ago patient took influenza and his first attack of bronchitis; has had bronchitis every winter but one of the last five; in hay season has sneezing fits sometimes for ten hours. Then, especially if there is also slight bronchitis, he is apt

to have severe asthma. Attacks are worst Sunday nights and Monday mornings; if he avoids food after midday on Sunday he has much less asthma on Mondays. The abdomen gets distended during attacks. In the mornings he is often "so hard up for breath" that he cannot put on his own boots. He gets his wife to do this, goes to work (6 a.m.), and in an hour is usually relieved; but once a week at least he cannot go till after 9 a.m. His first severe attack for three years was a fortnight ago, and lasted four days; he thought he was going to die of suffocation. Less severe attacks have often kept him off work for a week or two. There is slight emphysema, some cooing rales; much narrowing of left nostril, a spur on both sides of the septum, a hypertrophied, intensely irritable, sneeze-provoking area on the septum opposite to and touching each middle turbinal; lingual tonsil hypertrophied and irritating the epiglottis; larynx injected.

17th May, 1900. Patient regards himself as practically cured. For two years before treatment he had slight asthma every morning; now he is never breathless in the mornings, and has only thrice had asthma since treatment began—once after eating pastry—and none for three months. Has only twice lost a day's work. The glottis during one attack was noted to be widely dilated and still; cocain and suprarenal solutions to the interior of the nose, and especially to the base of tongue and epiglottis, on several occasions greatly relieved the asthma. Urine scanty during attacks; uric-acid in excess. The treatment has been pot. brom. and pot. iod. internally, with occasional doses of calomel; cauterization to the septum and lingual tonsil. Patient feels so well that he declines further nasal treatment.

CASE XXI.

Female, 22, tailoress, Hamilton. 27th March, 1900. I was first called to this case this evening, and found her very weak, but sitting up, breathing with extreme difficulty; face of a dusky pallor, extremities dusky, and pulse soft and feeble; heart rapid, tumultuous, and irregular; occasional spit of pink froth (mucus intimately mixed with blood); feeble

R.M.; small, moist, indistinct rales, but no cooing rales, all over lungs. The attack began yesterday (Monday), and increased so that she had to leave work at 5 p.m. Menstruation ceased on Saturday, and she had a bad headache that day and Sunday.

28th March. Mustard-bran poultices greatly relieved patient. Spit is no longer blood-stained, face and hands still rather dusky, abundant moist and cooing rales. Patient has been subject to asthma for two years, and had six attacks last summer, mostly on Sundays. She says the hands are always rather dusky, feet cold; no urticaria. The attack described made one think of acute capillary bronchitis, but was undoubtedly asthma, and was indistinguishable from the paroxysmal bronchitis of children, which, Gee says, *is* asthma. Temperature never rose above normal.

14th May, 1900. Both middle turbinals having been found hypertrophied and the right growing polypi, these were all treated; blue pill given on Saturdays and an iodide mixture during the week. There has been no more asthma.

P.S.—November, 1900. Patient has since had one or two attacks, relieved by paraldehyde.

CASE XXII.

Female, 50, Hamilton. 16th May, 1900. A thin, sallow woman, who weighs $6\frac{1}{2}$ stone in walking dress, under-eats, over-works, lives in a low, damp house, and has enough chloasma and general darkness of skin, but too much energy to make one think of Addison's disease. Asthma began two years ago, sometime after influenza and bronchitis, the first attack occurring after exertion, then becoming nocturnal, and latterly coming on after dinner. Flatulence, indigestion, and deterioration of the general health are present; also chronic pharyngitis, hypertrophy of lingual and faucial tonsils, hypertrophy of right middle turbinal, stenosis of left nostril owing to twist and spur of septum, which leans on the middle turbinal. Beyond cauterising lingual tonsil, treatment of upper respiratory tract was not pressed, as malnutrition is

evidently the chief factor, and difficult to remedy owing to patient's circumstances. Treatment of digestion has done most good, but asthma still troubles sufficiently to make patient burn asthma powder almost nightly.

P.S.—December, 1900. Since writing the above I have tried Dr. W. Murray's treatment with stramonium and arsenic. This was adopted two months ago, with complete disappearance of the asthma.

CASE XXIII.

Female, 39, Hamilton. 17th May, 1900. Bronchitis began when she was 15, and has recurred every winter save one, but latterly has been less severe. Asthma began, at least in severe form, seven years ago, but has not recurred since pneumonia, two years ago. Three or four severe attacks occurred yearly, lasting several days: they usually followed bronchitis, but rarely apart from bronchitis, and then after excitement. She thinks they were apt to occur at menstrual periods. Asthma occurred only at the beginning of the two last of her pregnancies. During attacks urine is loaded with urates. There is emphysema; nose and throat show nothing specially wrong. Cocain to interior of nose and amylnitrite gave a little relief. No marked benefit from mist. iod. et ars.

CASE XXIV.

A boy, aged 14, son of Case 23, had spasmodic croup when 6, bronchitis when 7, and asthma just when 8. Attacks are frequent, come in the mornings, and during the last two years mostly on Sundays and Mondays, so that he is rarely at work on Mondays. During these last two years he has rested more and eaten more on Sundays than during the week, and has played football on Saturdays. There is polypoid hypertrophy of both middle turbinals; and though patient does not snore in sleep, there are the little granules on the pharynx, suggestive of adenoids higher up. Left tonsil is enlarged. I have not been called to treat the asthma.

CASE XXV.

Female, 56. May, 1900. Patient was mother of Case 23, grandmother of Case 24. She had for years had chronic bronchitis before coming to Hamilton, and then for 12 years had asthma. She came to me nine years ago, and from that till her death, four years ago, from cardiac disease, considered herself cured. It is plain, however, that while she never had another real "attack" of asthma, she was afraid of one coming on, and often at bedtime took a dose of her medicine, *mist. iod. et ars.*, which she always kept in the house, and which was the chief treatment. No special abnormality was found in nose or throat.

ALBUMINURIA AND NEPHRITIS OCCURRING IN CONNECTION WITH SCARLET FEVER IN COMBINATION WITH VARICELLA, THE VARICELLA OCCURRING DURING THE ACUTE STAGE, OR DURING THE PERIOD OF CONVALESCENCE.

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WHEN acting as one of the assistant physicians in the City of Glasgow Fever Hospitals, at Belvidere and Kennedy Street, during the years 1893 and 1894, several outbreaks of chicken pox occurred amongst scarlet-fever patients, in the wards of which I had charge during the period referred to, occasioned chiefly by patients admitted incubating that disease. And of those so affected I remarked that decided albuminuria and acute nephritis occurred in a large number, and that in several instances the nephritis was markedly severe. Accordingly, I set myself the task of endeavouring to find out whether the occurrence of chicken pox, during the acute stage of scarlet fever or shortly thereafter, had any causative relationship to the occurrence of albuminuria or of nephritis, or tended in any way to aggravate such a condition when it already existed.

On looking into the literature of the subject I find no definite record of any inquiry into the question of the significance attaching to albuminuria and nephritis occurring in connection with scarlet fever in combination with chicken pox, although Henoch, who has placed on record four cases of chicken pox in which nephritis occurred as a complication, mentions that serious consequences may arise from the combination of varicella with other diseases—scarlet fever, measles, and diphtheria.

In virtue, however, of the great differences which scarlatinal

nephritis presents both as regards the relative frequency of its occurrence and as regards the degree of severity, it is a matter fraught with much difficulty to determine whether the disease superimposed, as it were, namely chicken pox, plays any part in determining the occurrence of albuminuria or nephritis, or in rendering what would otherwise be an attack of moderate severity one of grave import.

It will be fitting, therefore, in the first place to view the last-mentioned disease alone, in relation specially to the occurrence of albuminuria and nephritis.

Until recently chicken pox was regarded as a disease in which the prognosis was invariably favourable. And there can be little doubt that in the majority of instances chicken pox runs a comparatively mild course, and is seldom attended with danger; still, in the light of fresh accessions to our knowledge of this disease, it must be regarded as not so devoid of harm or even danger as it was formerly thought to be. A haemorrhagic form has been described, while pleurisy, secondary abscesses, and pyaemia have been noted as occurring in close association with it. And to these must be added nephritis, of which there are now quite a number of cases recorded. Particulars of a few are here given.

Henoch has placed four such cases on record. The important facts as gleaned from the account given by him are these: Oedema accompanied by albuminuria set in from eight to fourteen days after the appearance of the eruption of chicken pox. In most the eruption was copious, and accompanied by fever. In one, the youngest, a child aged two years, it proved fatal. In this case there was evidence of specific disease. In the others recovery took place in from fifteen to thirty-two days.

Rachel describes two such cases which came under his notice. In one, oedema of the feet and face were present during the eruptive stage, and the urine was moderately albuminous for twelve days. As it was thought that scarlet fever might be present, careful search was made for evidence of that disease, but none was found. In the second case, that of a child of six months, the eruption was confluent, especially over the chest and neck, there being also large patches over

the left buttock and sacrum. On the third day the vesicles showed signs of drying, and the febrile symptoms were subsiding. At this time it was noticed that very little urine was passed. The feet and hands were markedly swollen. On the sixth day some improvement took place as regards the oedema, but very little urine was passed. On the following day a specimen was obtained for examination, when it was found on boiling to contain a moderate amount of albumen. The quantity of urine passed was greater, and the oedema entirely gone. On the 14th day the urine was normal. This writer adds that possibly the severity of the attack had something to do with the occurrence of nephritis, as it was the only one in a series of seven cases of chicken pox under careful observation in which the eruption showed a tendency to become confluent. In the other six no signs of nephritis were found.

Similarly, other observers, such as Hoffmann, Rasch, Semtschenko, have put on record cases of nephritis occurring during the course of varicella. The last-mentioned observer found that the nephritis began, as in the second case narrated by Rachel, on the third day.

In accepting the testimony of these and other observers, therefore, we are constrained to assign an important place among the complications of varicella to nephritis. And according to the evidence before us this complication may show itself either during the first few days of the disease, as in the cases furnished by Rachel and Semtschenko, or at a later date, as in those by Henoch. It may also, in the case of unhealthy children, assume a severe or even a fatal form.

We must conclude then that a selective and baneful influence is, under certain circumstances, exercised on the kidneys in varicella, as in scarlet fever, diphtheria, and measles, although that influence may show itself more seldom.

Of the infectious diseases just mentioned, scarlet fever is the one in which nephritis holds the chief place as a complication or sequela, in virtue of its relative frequency and significance. And one of the principal features of the scarlatinal process, to the significance of which much attention has been devoted, is the presence of albumen in the urine. It

may appear in such small amount as to be just perceptible as a faint cloud on applying the heat test with acetic acid, or the whole column may quickly coagulate. All gradations are found between these two extremes. As is well known, however, a trace of albumen sometimes appears in the urine during the febrile state, and more particularly when the temperature reaches a high level. It is, however, at most transitory, disappearing in the course of a day or two with the receding temperature. Still, when it occurs in connection with scarlet fever, it may be a matter of extreme difficulty or even impossibility to say whether or not the albuminuria is to be attributed to the pyrexial disturbance, or to certain changes in the kidneys incidental to scarlet fever. For thus early in the course of the disease—that is to say during the first few days or during the acute stage—albumen, sufficient only to show a faint or deep cloud, may appear in the urine and remain present for several days, while there may be no symptoms or signs besides this referable to the kidneys. Inasmuch, however, as the albumen may suddenly become greater in amount, and blood suddenly appear, or a more gradual increase in the amount of the albumen take place, and inasmuch as symptoms indicative of pronounced renal changes, such as pallor of the face and lips, puffiness under the eyes, restlessness, sickness and vomiting, may set in coincidentally, I think we are warranted in regarding these slight forms of albuminuria in which only a very small amount of albumen is regularly detected for a short period, as representing certain departures from the normal renal condition, differing only in degree from those found in the more severe forms of the renal affection. I am of opinion that we must look upon this condition, *i.e.* albuminuria, whether occurring late or early in the course of the disease, as implying certain changes in the kidneys other than functional disturbances.

Support is added to this view by the fact that at a later stage—that is to say during the third week, and for several weeks thereafter, the period during which the severe affection of the kidneys most frequently occurs—a trace of albumen may be present on two or three occasions in succession, and disappear, to reappear once or oftener for brief periods during

the time that the patient is in hospital. Moreover, albumen, in larger amount, or blood may suddenly make its appearance, and after one or perhaps two days either or both may as suddenly disappear, only to reappear abruptly after a lapse of some days, and after remaining present for as brief a period may again disappear as abruptly as before. And it is necessary in this regard not to lose sight of the fact that transitory forms of slight albuminuria occur more frequently during health than was at one time believed. Consequently it is natural to conclude that they have to be reckoned with when considering the question of the relative importance of albuminuria in scarlet fever, as also in chicken pox.

Again, it is a fact worthy of note that scarlatinal nephritis may exist without any albumen being detected in the urine, although careful search has been made; or it may be detected at one period and subsequent examination may fail to reveal its presence, and yet when examined post-mortem the kidneys may present the appearances peculiar to genuine nephritis of scarlatinal origin.

It is necessary before proceeding further to explain clearly what we mean by the terms albuminuria and nephritis. They refer to clinical manifestations, and are arbitrarily employed for the purpose of grouping the cases observed into categories, and instituting such comparisons and making such deductions as can properly be formed. The term albuminuria is used simply to denote the presence of albumen in the urine. When it is associated with blood or blood-colouring matter, or when definite symptoms and signs referable to the kidneys are present in addition to the albumen, the term nephritis is employed. But for the reasons specified above, we are strongly disposed to believe that in both the pathological condition is essentially of the same nature, and that "decided" as distinguished from "transitory" albuminuria implies definite, if slighter, changes in the organs in question.

The different views held as to the relationship existing between the presence of albumen in the urine and the actual nephritic process have, I believe, much to do with the widely different results arrived at by different observers as to the frequency and significance of scarlatinal albuminuria and nephritis.

Other important factors are the character of the epidemic, the particular mode, as also the regularity and frequency of testing the urine. At the same time much must, no doubt, be attributed to the skill and care of the individual observer. In illustration of this, the following results are given in tabulated form :

Total number of Cases observed.	Albuminuria and Nephritis occurred in	Authority.	REMARKS.
5443	8.47 per cent.	Goodall	Albumen present on more than one occasion, and not febrile
4015	7.69 ,,	Caiger	
365	20 ,,	Sørensen	
180	62.2 ,,	R. S. Thomson	
91	52.7 ,,	F. Dittmar	

It is evident from a consideration of the above that comparisons are of little practical value in the absence of a common basis, and in my investigations I have had regard to this. I first sought to find out the proportion of cases of scarlet fever in which albuminuria and nephritis occurred during a given period. I next proceeded to find out the proportion of cases of scarlet fever and chicken pox in combination, in which albuminuria and nephritis occurred during the same period; and in the third place to compare the results so obtained. And in this connection it is to be noted that the cases occurred during the same epidemics, and that, as far as possible, the same conditions as regards diet and treatment obtained throughout; also, that uniform frequency was maintained in the examination of the urine. In each case it was tested morning and evening during the first week, or until the temperature became normal, then once daily till the end of the third week, the specimen being obtained as a rule in the morning. Afterwards the urine was examined every alternate day till dismissal, unless albumen or blood or blood-colouring matter was detected, or other indication of nephritis showed itself, in which case the examination

of the urine was again conducted morning and evening till the disappearance of the albumen, and at least once daily for a week thereafter. In those cases in which chicken pox occurred in addition to scarlet fever, the urine was examined morning and evening while the eruption was present, and once daily after the vesicles had dried until the crusts had separated. In a large number of the cases of scarlet fever, both attended and unattended with nephritis, the examination of the urine was undertaken personally, while in those in which chicken pox occurred in addition, special attention was devoted to the examination of the urine. In a number of instances the urinary sediment was examined microscopically. The number of patients, however, in the City of Glasgow Fever Hospitals at Belvidere and Kennedy Street suffering from scarlet fever during the periods referred to, viz. the years 1893 and 1894, were greatly in excess of any previous year, and this made it well-nigh impossible to make such an examination as systematic or as thorough as one would have desired.

In testing for the presence of albumen, the method practised was as follows: The reaction was taken, then the cold test with nitric acid was applied as being the one most expeditiously carried out, care being taken, however, to prevent the co-mingling of the two fluids at the plane of contact. In addition to this the heat test with dilute acetic acid, was frequently combined, and if there seemed to be any fallacy or any doubt as to the result, the picric acid test was resorted to. While for the purpose of detecting blood and blood-colouring matter, the guaiac test and the microscope were employed.

In the matter of food uniformity was maintained. In the acute stage the diet was limited to milk and farinaceous food; no butcher meat was given till after the end of the fourth week; during convalescence chicken soup, beef tea, potatoes and mince, chicken, fish, and vegetables were allowed. Should, however, albumen make its appearance in the urine during this stage, patients were forthwith put on milk and a farinaceous diet. The medicinal treatment followed was practically the same throughout and consisted of purgatives, diuretics,

linseed poultices to the loins, packs; in severe cases with scanty urine, dry cupping; in severe and protracted cases, to avert the supervention of uraemic symptoms, nitro-glycerine was found to be of benefit; when uraemic symptoms occurred, chloral suppositories with the administration of chloroform.

All the cases enumerated below, including those in which chicken pox occurred in combination with scarlet fever, were, with one or two exceptions, under my care till dismissal. They are all consecutive. The investigation is based on cases in which chicken pox appeared concurrently with the acute stage of scarlet fever, or during the period between the termination of the acute stage and that of the residence of the patient in hospital. The cases observed at Belvidere Hospital and those at Kennedy Street are taken separately. Those in which albuminuria in the slightest degree occurred on more than two occasions, together with those in which it was more decided, have been placed in the same group with those in which pronounced symptoms and signs of renal mischief showed themselves, for the reasons already given.

Cases in Belvidere Hospital.

Scarlet fever, irrespective of the occurrence of chicken pox,	476
From that number there fall to be deducted : children $1\frac{1}{2}$ years and under in which the urine was not obtained for examination,	11
Cases of unusual severity in which the urine was passed involuntarily,	3
Cases in which chicken pox occurred,	38
	<hr/> 52
	<hr/> 424

Of these 424 cases, 67 presented signs of albuminuria and nephritis, that is 15·80 per cent. While of the 38 cases in which chicken pox occurred, 8 presented signs of albuminuria and nephritis, viz., 21·05 per cent.

Cases in Kennedy Street Hospital.

Scarlet fever, irrespective of the occurrence of chicken pox,	924
From that number there fall to be deducted: children $1\frac{1}{2}$ years and under in which the urine was not obtained for examination,	7
Cases of unusual severity in which the urine was passed involuntarily,	15
Cases in which chicken pox occurred,	14
	— — 36
	888

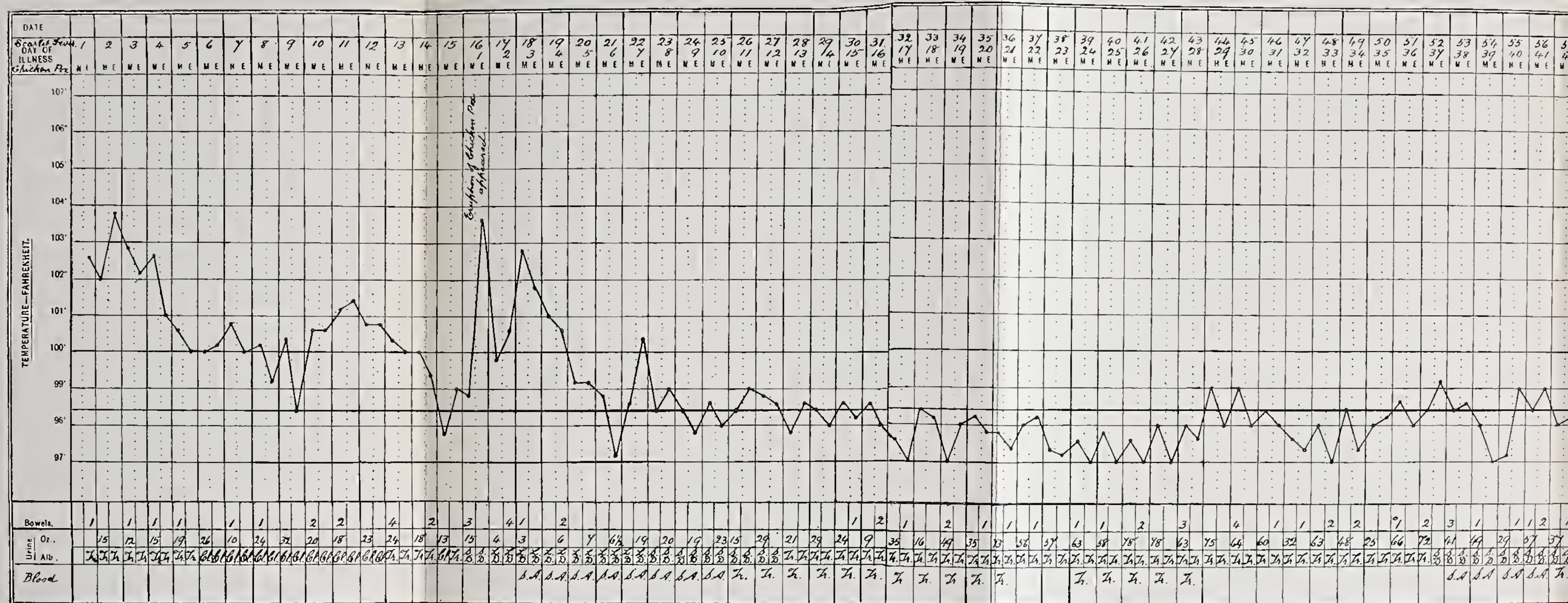
Of these 888 cases, 125 presented signs of albuminuria and nephritis, or 14·07 per cent. While of the 14 cases in which chicken pox occurred, 4 presented evidence of albuminuria and nephritis, or 28·27 per cent.

It will be seen that at both hospitals the percentage of instances in which albuminuria and nephritis occurred in association with chicken pox is decidedly greater than that in which the same complication or sequela occurred in connection with scarlet fever alone. Still the number of cases observed in which the exanthemata referred to co-existed is limited, and on that account I do not seek to attach more importance than may legitimately be done to this purely statistical part of the investigation. It were well to consider along with it the principal features of the individual cases in which scarlet fever and chicken pox co-existed, and in which albuminuria or nephritis occurred, and to submit them to examination and analysis. They are taken consecutively and in chronological order.

CASE I. (See Chart.)

J. R., aet. $6\frac{1}{2}$. Admitted on 1st day of illness. Rash distinct, generalised. Fauces reddened; tumefaction of soft palate. Trace of albumen in urine.

16th day. Trace of albumen present for first five days, then on 13th and 14th days. This afternoon began to complain of headache; evening temperature, $103\cdot6^{\circ}$ F. Only 15 oz. urine passed during last 12 hours. It was dark red



SERIES I., CASE I.



in colour, and contained albumen and blood in considerable amount. Copious sediment consisting of urates, granular debris, red corpuscles, and epithelial and hyaline casts. Several clear vesicles appeared on head and chest.

17th day. Condition of throat much improved. Was sick during the night, when only 2 oz. of urine were passed. No oedema or dropsy.

18th day. Urine still very scanty; small amount of blood present and considerable amount of albumen.

19th day. Was sick and vomited once during the day; on the whole has been more restful. Slight oedema of the face; lower eyelids not markedly involved; no oedema seen elsewhere. Urine contains less blood; still very scanty and highly albuminous.

20th day. Was sick once; bowels moved freely; large quantity of urine passed during the day; temperature sub-normal; pulse 80, of high tension. Right ear has begun to discharge to-day.

22nd day. No sickness; 19 oz. urine passed during the last 24 hours; still contains a small amount of blood; it is smoky and of slight red tint. Discharge from ear is less.

23rd day. No sickness; 23 oz. urine passed in last 24 hours; still smoky; no red tint; amount of albumen less.

24th day. Free from sickness; 23 oz. urine passed; smoky; again red; amount of albumen same. Pulse 80, of high tension; irregular.

25th day. Pallor of face and puffiness below eyes observed. Blood and albumen present; albumen less in amount and corresponds almost to the amount of blood present.

26th day. Urine passed this evening straw-coloured; responded slowly to the test for blood.

27th day. Temperature normal, and frequently sub-normal; pulse irregular. 41 oz. urine passed in 24 hours. Blood and albumen same in amount as on previous day.

29th day. 60 oz. urine passed; has slight smoky appearance. Pulse 65, irregular; no systolic murmur heard. Several vesicles on head and chest have become pustular and are surrounded by hyperaemic areas.

31st day. Condition of urine same. Oedema of face and

puffiness below the eyes still present. Pulse 65, irregular, less hard. A soft ventricular-systolic murmur heard at apex and mid-sternum; loudest in the latter situation. Second aortic sound accentuated.

36th day. Quantity of urine has been increasing, 73 oz. passed in last 24 hours; no response to the guaiac test; albumen very small in amount. Systolic murmur scarcely audible; pulse more regular.

43rd day. Upwards of 60 oz. urine passed in 24 hours; blood can just be detected; amount of albumen small. Heart sounds pure. The pallor has disappeared from the face and also the oedema.

52nd day. Improvement continued; 70 oz. urine passed daily; blood has been absent for more than a week; a trace of albumen remains.

57th day. Urine abundant; blood present in small amount from 53rd day with slightly greater amount of albumen than would be accounted for by the presence of blood alone.

95th day. Has been improving steadily. Entire absence of albumen since the 60th day. Recovery complete; dismissed.

CASE II.

R. R., aet. 4½. Admitted on 1st day of illness. Rash distinct on chest, abdomen, arms, and legs, faint elsewhere. Tongue coated with a brownish-white fur; fauces deeply injected; tonsils enlarged, bright red, accumulation of yellowish deposit on the surface of each. Sub-maxillary gland on right side enlarged. Temperature on admission, 101° F. Heart sounds pure, and normal in rhythm. Urine contains urates in abundance; a faint trace of albumen also detected.

45th day. Has pursued a mild course; no albumen present except a faint trace on admission. Was convalescing satisfactorily and out of bed. An eruption of papules and vesicles appeared to-day on the head and face, as also on chest and back; no red areolae or erythematous blush present. Morning temperature, normal; evening temperature, 100° F. The urine contains blood in small amount and albumen in larger amount than is to be accounted for by the amount of blood.

46th day. Morning temperature, 99° F. A few fresh vesicles seen in the same situations; eruption not abundant. The face is paler than before; no oedema or dropsy present; no diminution in the quantity of urine; small amount of blood: albumen sufficient to form a deposit.

53rd day. The vesicles have dried, and the crusts have nearly all separated. Until the 51st day a small amount of blood was present. Albumen in small amount on this and on the succeeding day detected.

63rd day. The urine has been free from albumen since the 52nd day. Completely recovered; dismissed.

CASE III.

M. M., aet. 4 Admitted on the 10th day of illness. History of sickness and sore throat 9 days ago; a swelling noticed in neck 6 days ago. Dry papular condition of skin, especially of the extremities. Fauces red and soft palate oedematous; tonsils enlarged, more especially the left; dirty white fur on tongue; slight bilateral enlargement of glands at angles of jaw; cervical glands on left side also enlarged, and neck has a brawny feeling for some distance around the enlarged glands. Temperature on admission 101·8° F. Urine free from albumen.

11th day. Deep fluctuation in swelling on left side of neck. An incision was made and exit given to a quantity of pus and serous fluid. Drainage was established and the wound dressed antiseptically.

18th day. Cavity in neck filling up and wound draining satisfactorily. Temperature last evening was 102° F.; this morning 99° F. Quantity of urine passed in last twenty-four hours small, only 7½ ounces. Blood present in small amount and albumen in larger amount than may be accounted for by the blood.

22nd day. An eruption consisting of papules and vesicles has appeared. Its characters are those of chicken pox. The febrile disturbance is slight. The albumen is greater in amount, while the blood is the same. The urine is very scanty.

24th day. The urine continues to be scanty ; amount of albumen very considerable ; blood stationary in amount.

54th day. Steady improvement has been taking place since last note was made ; the urine is normal in quantity. A trace of albumen remained until three days ago. The urine is now free from albumen and blood. Wound almost healed.

68th day. Completely recovered ; dismissed.

CASE IV.

C. M., aet. 4. Admitted on the 4th day of illness. Fading rash with distinctive characters seen, and also a dry papular condition on trunk. Reddening of fauces with oedema of uvula and soft palate. Tongue moist, slightly coated ; presents "strawberry" characters. Slight bilateral enlargement of sub-maxillary glands. Temperature on admission 101.2° F. Pulse soft and rapid.

8th day. The temperature keeps above 120° F. ; restless ; has a sunken aspect ; profuse muco-purulent secretion from tonsils and posterior wall of pharynx, also yellowish deposits seen on both tonsils. Purulent discharge from nostrils. A large number of papules and vesicles are appearing on face, trunk, and limbs. A trace of albumen was present in the urine yesterday.

9th day. Patient becoming weaker ; glands on both sides of neck becoming hard and brawny ; no deep fluctuation can be detected. Throat, dusky red ; secretion more viscid ; discharge from nostrils offensive ; pulse feeble and rapid. A fresh crop of vesicles seen ; those of earlier crop are drying in ; eruption copious ; red areolae round many of the vesicles. Since the 7th day of illness the urine has been passed involuntarily.

16th day. Patient's condition is becoming worse, although the temperature remains within moderate limits, ranging between 100° and 102.2° F., with morning remissions ; it was previously inverted. Pulse small, soft, and rapid. Specimen of urine obtained to-day ; albumen present in large amount ; no blood detected.

19th day. Weakness increasing; temperature has been rising higher and higher since yesterday.

20th day. Died at midnight; two hours before death the temperature was 108° F.

CASE V.

R. P., aet. 6. Admitted on 2nd day of illness. Diffuse blush on trunk and legs; red mottling on forearms. Fauces reddened; both tonsils enlarged, left much more so than right. Creamy fur on tongue. Submaxillary gland on left side enlarged. Urine contains albumen sufficient in amount to form a deposit. Temperature, 103.2° F.

8th day. The temperature has been normal since the 3rd day. Upwards of 40 oz. urine passed daily; a trace of albumen has been present continuously from the 2nd day. No pallor of face nor oedema of feet or of face.

14th day. Faint trace of albumen for two days, alternating with absence of albumen for a like period.

28th day. Submaxillary glands enlarged and painful; trace of albumen present; no blood.

44th day. A trace of albumen for one or two days, alternating as before with absence of albumen for several days until to-day, when albumen was present in larger amount; no sickness; no blood; no oedema or dropsy.

56th day. Urine free from albumen on 54th and 55th days. Numerous red papules and vesicles on face, trunk, and legs. Constitutional disturbance not marked. Urine found to be again highly albuminous; no blood present.

78th day. Urine now free from albumen, and has been so for ten days. All crusts have separated. Completely recovered; dismissed.

CASE VI.

J. B., aet. 7. Admitted on 2nd day of illness. Moderately bright rash seen on trunk, arms, and legs. Bilateral enlargement of submaxillary glands with tenderness. Fauces reddened; tumefaction of soft palate, and pillars of fauces;

tonsils much swollen. Tongue glazed and rather dry. Urine contains a trace of albumen.

8th day. The urine contained a trace of albumen each day till to-day, when it was clear.

22nd day. The temperature, which has been almost normal during the past eight days, rose this evening to 100·8° F. No complaint of sickness. The urine contains a deposit of albumen; also blood in small amount.

40th day. Albumen sufficient to form a deposit, and blood in small amount present till the 37th day. On the 37th and 38th days blood was absent and the albumen smaller in amount. On the 39th day only a trace of albumen was present. It was clear to-day.

45th day. The urine has remained clear since the last note was made.

55th day. A number of isolated papules and vesicles having the characters of the eruption of chicken pox appeared on the face, chest, abdomen, and legs. The temperature rose this evening to 101° F. Albumen returned in small amount; no blood present.

77th day. The eruption was moderately copious; the crusts have now separated. The albumen remained present for 4 days; since then the urine has been clear. Completely recovered; dismissed.

CASE VII.

D. F., aet. 4. Admitted on 2nd day of illness. Moderately bright rash, punctate, seen on arms, legs, chest, and abdomen. Fauces bright red; slight oedematous swelling of soft palate and uvula; tongue cleaning; urine clear.

6th day. Urine clear till to-day, when it was found to contain blood and albumen, the former in small amount, the latter sufficient to form a deposit; slight diminution in the quantity of urine.

13th day. Albumen still present in small amount; no blood detected.

27th day. The quantity of urine has been increasing; no blood has been present, but albumen in the form of a trace for two or three days at a time, on more than one occasion.

The temperature has been above 102° F. for the last two evenings, and to-day an eruption of vesicles on the chest, abdomen, arms, and legs appeared. Albumen has reappeared in sufficient amount to form a deposit; no blood detected.

56th day. Eruption was moderately copious; the crusts have now separated. Albumen present during five days in succession, and in decreasing amount; since then the urine has remained clear. Completely recovered; dismissed.

CASE VIII.

E. S., aet. 8. Admitted on 2nd day of illness. Moderately bright rash, diffuse, seen on limbs and trunk. Hyperaemia of fauces; moderate oedematous swelling of uvula and soft palate; tonsils swollen and lobulated; tongue moist, clean at tip; papillae prominent; submaxillary gland on each side enlarged; urine clear.

27th day. Course has previously been mild and uneventful. A number of small red papules and oval and rounded vesicles have appeared on the legs and anterior aspect of the body and left side of the face; an erythematous blush on chest, abdomen, and back seen. The urine contains a small deposit of albumen, but no blood.

28th day. A few vesicles on the back are beginning to dry, while fresh ones are appearing; 24 oz. urine passed in last 24 hours; amount of albumen remains the same.

56th day. Two days after last note was made only a trace of albumen was present in the urine; this remained during the two following days, after which it was clear. Completely recovered; dismissed.

CASE IX.

M. M'A., aet. 6. Admitted on the 4th day of illness. Rash well defined, discrete, and moderately bright; well out on chest and arms; uvula and soft palate oedematous; both tonsils swollen; accumulation of muco-purulent secretion in pharynx; tongue moist; anterior half studded with swollen papillae; submaxillary gland on left side enlarged and tender; temperature on admission 101·8 F.; urine clear.

6th day. The range of temperature has been moderate, the highest temperature being 101.8° F. This evening it has risen to 103.4° F. An eruption of minute, red papules and vesicles has appeared to-day; they are not numerous, and are situated on the face, chest, and abdomen. The urine is moderate in quantity and free from albumen.

8th day. A fresh crop of vesicles has appeared to-day, principally on the chest and abdomen; the others are drying. No albumen present.

10th day. The temperature is descending; a trace of albumen was present in the urine morning and evening.

11th day. Albumen, sufficient in amount to form a deposit, was present this morning; in the evening it amounted to a trace.

24th day. The urine has now been clear for a week; one or two crusts still adherent.

56th day. All crusts have separated. Completely recovered; dismissed.

CASE X.

M. C., aet. $5\frac{1}{2}$. Admitted on the 2nd day of illness. Discrete well-marked rash seen on chest, abdomen, legs, and arms; there are also a few pustules seen, together with several irregularly-rounded crusts and cicatrices, on the head, trunk, and limbs. They present the characters of a fading varicellar eruption. Fauces are reddened; both tonsils swollen; accumulation of pappy deposit on each. Submaxillary lymphatic glands are enlarged and tender. Urine clear.

13th day. Temperature during the first few days was moderately high; it did not exceed 102.4° F., and came down to normal on the 8th day. The urine was clear until the 11th day. Some of the crusts have separated; others are drying. The urine is not diminished in quantity; it has contained a trace of albumen morning and evening since the 11th day.

17th day. A trace of albumen persisted till yesterday. The urine is now free from albumen.

57th day. It has remained clear, and progress has been uninterrupted; dismissed.

CASE XI. (*See Chart.*)

J. S., aet. 4. Admitted on the 3rd day of illness. Bright confluent rash, generalised. Hyperaemia of fauces; no oedema of uvula or soft palate. Tongue moist, presents "strawberry" characters. The urine contains a trace of albumen.

30th day. A trace of albumen was likewise present on the morning and evening of the 4th day; the urine has been clear since then; and on the 3rd and 4th days the evening temperature exceeded 103° F. Afterwards the course was uneventful, and patient was allowed up to-day.

45th day. The temperature became elevated this evening, rising to 102·4° F. A number of red papules and vesicles appeared to-day; some of the latter are rounded and others elliptical or oval, and stand out of the healthy skin like beads; the eruption is characteristic of varicella. Urine found to be clear.

46th day. A few fresh vesicles seen to-day, principally on chest and abdomen; the temperature is not elevated. The urine is still clear.

48th day. Groups of vesicles drying and fresh ones appearing. The temperature has risen this evening to 103° F. A very considerable amount of albumen has appeared in the urine; coagulation of almost the whole column takes place on boiling and adding acetic acid. No blood is detected. The quantity of urine is not diminished.

52nd day. Patient was sick during the day and vomited once. Still no diminution in the quantity of the urine; it has a crimson tint. Blood is present in considerable amount and there is a very large amount of albumen. Microscopic examination of the sediment revealed the presence of numerous red blood corpuscles, renal epithelial cells, hyaline tube-casts and granular debris.

55th day. Quantity of urine passed distinctly smaller, viz., 19 oz. in the last 24 hours; it is dark red in colour. The albumen is still considerable in amount ($\frac{1}{2}$ column). Was sick and vomited several times during the night and day.

60th day. A larger quantity of urine passed—39 oz.

There has been no blood present for 3 days. The amount of albumen is smaller ($\frac{1}{8}$ column). It has also been clear on three occasions, and there has been a trace of albumen on two occasions. A sufficient quantity is passed.

88th day. Further improvement in the condition of the urine observed; it is clear this evening; not more than a trace of albumen morning and evening since the 84th day.

102nd day. A much larger quantity of urine passed; has exceeded 70 oz. With the exception of one occasion, it has been clear morning and evening for a week.

120th day. The urine has been entirely free from albumen for upwards of 10 days. Completely recovered; dismissed.

CASE XII.

J. H., aet. $5\frac{3}{12}$. Admitted on third day of illness. Moderately bright rash, discrete, on chest and arms. Fauces reddened; moderate degree of tumefaction of uvula and soft palate; no deposit on tonsils. Tongue clean, bright red; papillae prominent. Submaxillary glands on both sides slightly enlarged. Urine clear.

10th day. The temperature became normal on the 6th day of illness; last evening it rose to 101.2° F. Does not complain of pain or discomfort.

11th day. An eruption observed this morning on trunk, legs, and arms, and slightly on face, consisting of a considerable number of vesicles and a few minute red papules. The eruption is typical of chicken pox. No sickness nor headache. Urine clear.

15th day. Fresh crops have been appearing daily; the eruption is copious and semi-confluent; some of the vesicles are large, and flattened, and umbilicated. The temperature has exceeded 103° F. Urine has been entirely free from albumen until to-day, when it appeared in considerable amount. A small quantity of blood is present.

24th day. Many of the vesicles have become pustular; an erythematous rash is seen, especially on the posterior aspect of the trunk. The temperature has descended and remained low till this evening, when it mounted quickly to 105.4° F. The



amount of albumen has increased to $\frac{1}{8}$ column; the amount of blood present is also greater.

31st day. Slight improvement in the condition of the urine; the albumen is less and also the blood. The temperature continues to run high, ranging between 103° and 104° F. Pulse 120; tension not high. Has vomited frequently; skin moist.

34th day. Weakness increasing; pulse 120, soft and irregular. Respirations frequent and shallow, 40. Dulness in infra-scapular and lower axillary regions, where the R.M. is markedly defective. Has diarrhoea, the stools being yellowish and watery. Was very restless, and also delirious during the night and very excited during the day. His vision became impaired; he spoke of seeing only one half of certain objects; and when one of the nurses was standing at the foot of his bed he said he only saw one half of her body, namely, the right. He had orthopnoea; there was also general anasarca. Patient became more prostrate and the urine became worse. It had a deep red colour from the large amount of blood present, and there was a notable increase in the amount of the albumen; tube-casts were present in abundance.

35th day. Became comatose; previous to this squinting of the left eye was observed, the eye being rotated outwards. Died the same evening, without regaining consciousness.

It will facilitate the examination and analysis of the cases narrated, more or less in detail, if the main facts connected with them are presented in a tabulated form :

[Table

No. of Case.	Albuminuria or Nephritis before onset of Varicella.	Day of illness of Varicella appeared.	Day of illness of Scarlatina on which Nephritis occurred.	Day of illness of Varicella on which Albuminuria or Nephritis occurred.	Nature and Duration of Albuminuria or Nephritis, after onset of Varicella (day of illness of Scarlatina).	Character of Varicellar attack.	Result.
1	Albuminuria, 1st to 5th and 13th to 14th	16th	16th	1st	Nephritis, severe, 16th to 60th day	Eruption moderately copious, Febrile disturbance moderate	Recovered completely
2	Neither	45th	45th	1st	Nephritis, moderately severe, 45th to 52nd day	Eruption moderately copious, Febrile disturbance slight	Recovered completely
3	Nephritis from 15th day	22nd	22nd	—	Nephritis, severe, 18th to 50th day, increase in severity after 22nd day	Eruption moderately copious, Febrile disturbance slight	Recovered completely
4	Neither	8th	8th	4th	Nephritis, severe, from 12th day onwards	Eruption copious, Febrile disturbance severe	Died
5	Albuminuria, 2nd to 8th and 44th to 54th	56th	56th	1st	Albuminuria, decided, 56th to 68th day	Eruption copious, Febrile disturbance moderate	Recovered completely
6	Albuminuria, 1st to 8th; Nephritis, 22nd to 40th	55th	55th	1st	Albuminuria, slight, 55th to 59th day	Eruption moderately copious, Febrile disturbance moderate	Recovered completely
7	Nephritis, 6th to 24th	27th	27th	1st	Albuminuria, decided, 27th to 32nd day	Eruption moderately copious, Febrile disturbance moderate	Recovered completely
8	Neither	27th	27th	1st	Albuminuria, decided, 27th to 31st day	Eruption moderately copious, Febrile disturbance slight	Recovered completely
9	Neither	6th	9th	3rd	Albuminuria, decided, 9th to 17th day	Eruption moderately copious, Febrile disturbance severe	Recovered completely
10	Neither	1st	11th	11th	Albuminuria, slight, 11th to 16th day	Eruption moderately copious, Febrile disturbance moderate	Recovered completely
11	Neither	45th	48th	3rd	Nephritis, severe and protracted, 48th to 95th day	Eruption copious, Febrile disturbance severe	Recovered completely
12	Neither	11th	15th	4th	Nephritis, very severe, 15th day onwards	Eruption very copious, Febrile disturbance severe	Died

TABLE SHOWING MORE PARTICULARLY THE RELATION
OF ALBUMINURIA AND NEPHRITIS TO THE
OCCURRENCE OF CHICKEN POX.

12	Those in which Albuminuria or Nephritis occurred for the first time coincidently with or shortly after the onset of Chicken Pox.	7	After onset of Chicken Pox.	Nephritis: died. 2 Nephritis, attack severe and protracted. 1 Nephritis, attack moderately severe. 1 Albuminuria, decided. 2 Albuminuria, slight. 1
	Those in which Albuminuria or Nephritis occurred prior to the onset of Chicken Pox.	5	Nephritis prior to onset of Chicken Pox. 3 Albuminuria prior to onset of Chicken Pox. 2	Distinct accentuation of symptoms and signs of Nephritis. 1 Decided Albuminuria after urine had been entirely free from albumen for 3 days. 1 Slight Albuminuria after urine had been entirely free from albumen for 15 days. 1 Nephritis severe after urine had been entirely free from albumen for 2 days. 1 Decided Albuminuria after urine had been entirely free from albumen for 2 days. 1

It will be seen from the foregoing tables that the albuminuria and nephritis occurred most frequently on the 1st day of illness of varicella, or the day on which the eruption appeared, and with equal frequency on the 3rd and 4th days. In one instance the albuminuria was later in occurring, viz., on the 11th day.

Then with regard to the nature of the varicellar attack, in 5 cases it was severe, in 5 moderately severe, and in the remaining 2 it was mild in character. In none, however, could the eruption be said to be scanty. It was most frequently moderately copious, viz., in 8 cases. In 3 it was copious and in 1 very profuse.

All the above cases, except two, recovered completely. Both of the latter proved fatal. In each case a *post-mortem* examination was sought, but was not obtained. In one there would seem to be little doubt, from the very pronounced symptoms

referable to the kidneys and the condition of the urine, that the fatal issue was brought about by the intensity of the nephritic process; for, as will have been observed, there was oedema and anasarca, with, in all probability, dropsy of the pleural cavities, while the urine was largely composed of blood. And it is worthy of note that the varicellar attack was markedly severe. The constitutional disturbance was pronounced, and the eruption very copious and partially confluent. Many of the vesicles were large, and became pustular and also umbilicated. In the other case the cause of death is not so manifest. The facts as they stand are insufficient to enable us to form an opinion as to whether chicken pox may be regarded as having contributed to bring about the fatal issue. It is more probable, however, considering all the diagnostic features, that a general septic infection took place, as the attack of scarlatina conformed to the "anginose" type. At the same time it is to be noted that the varicellar eruption was copious and appeared early, and that evidence of nephritis was present.

Reference may be made here to certain points, in connection with the preceding cases, of more than usual importance. In the nephritis peculiar to scarlet fever as it is seen in hospital practice, oedema, in the experience of most recent observers, is not of frequent occurrence. And my own experience accords with this. Nor was oedema a notable feature of the cases under consideration as regards the frequency of its occurrence, although in one case it was present in a very marked form. It was met with altogether in two cases—numbers 1 and 12. In the former there was a moderate degree of oedema of the face and puffiness below the lower eyelids; there was no oedema elsewhere. In the latter, oedema of the face was present early, and to a marked extent; and at a later stage a condition of anasarca showed itself, with probably also fluid in both pleural cavities, judging by the degree of dulness and resistance in both infra-scapular and lower lateral regions and the marked deficiency of the respiratory murmur in these regions. The face and anterior and posterior aspects of the trunk were studded with vesicles and pustules, some of which were of considerable size; an erythematous rash was also

present on the anterior aspect of the abdomen and chest, and also on the posterior aspect of the body in the lower dorsal and lumbar regions. The oedema was in part at least to be accounted for by the numerous lesions in the skin; it was, in short, a "serous saturation" of the lymphatic channels and spaces of the subcutaneous cellular tissue.

Certain striking phenomena indicative of the uraemic state likewise occurred in this case, and are worthy of note, inasmuch as they are but rarely met with collectively as they are here. The uraemic condition was ushered in by a state of nervous irritability: the boy's temperament underwent a complete change—from being quiet and docile he became irritable and obstinate. His intellect remained clear until the onset of coma. On the day preceding that on which he died squinting of the left eye was observed; it was directed outwards. The strabismus remained present for several hours, when the normal direction was resumed. The patient also spoke of not seeing properly, and on making an examination it was found that there was a loss of vision limited approximately to the right half of the visual field; the defect was therefore binocular. No ophthalmoscopic examination was made. These phenomena are partly, no doubt, to be explained by the action of toxic products on the central nervous system in causing an excitation of the centres involved, but without further data we are unable to say whether any other factors, such as oedema or anaemia of the brain, played any part in the causation of these phenomena.

To return to the question at issue. It has been viewed from a statistical standpoint, and having considered the pertinent features of the individual cases in which both diseases co-existed, it remains to proceed to draw what conclusion may properly be formed from a consideration of the foregoing premises. It has been shown that albuminuria and nephritis occurred very frequently, immediately or very soon after the appearance of the varicellar eruption, or that a return of the albuminuria or nephritis where it existed previously, or a decided accentuation of either, took place coincidently with the onset of varicella. These facts, together with the preponderance at both hospitals of the cases in which albuminuria

and nephritis occurred (when both diseases co-existed) over those in which either complication appeared in connection with scarlet fever alone, go to show that some influence operating deleteriously on the kidneys was superadded, with the incidence of chicken pox.

Without, however, dwelling further at this point on the result arrived at, I would proceed to cite a further series of cases which occurred in the course of two slight outbreaks of chicken pox amongst patients suffering from scarlet fever in Kennedy Street Hospital, the one commencing in November, 1897, and the other in May, 1898. The former was limited to six cases; the latter comprised twelve. Special attention was paid to the examination of the urine during those outbreaks. During the one which began in November, one patient developed albuminuria in a decided form within a few days after the appearance of the eruption of chicken pox. During the outbreak which began in May, two such cases occurred, one of nephritis distinctly accentuated after the onset of chicken pox, and one of albuminuria several days after the appearance of the characteristic eruption of that exanthema.

Notes of these cases are subjoined.

SERIES II. CASE I. (See Chart.)

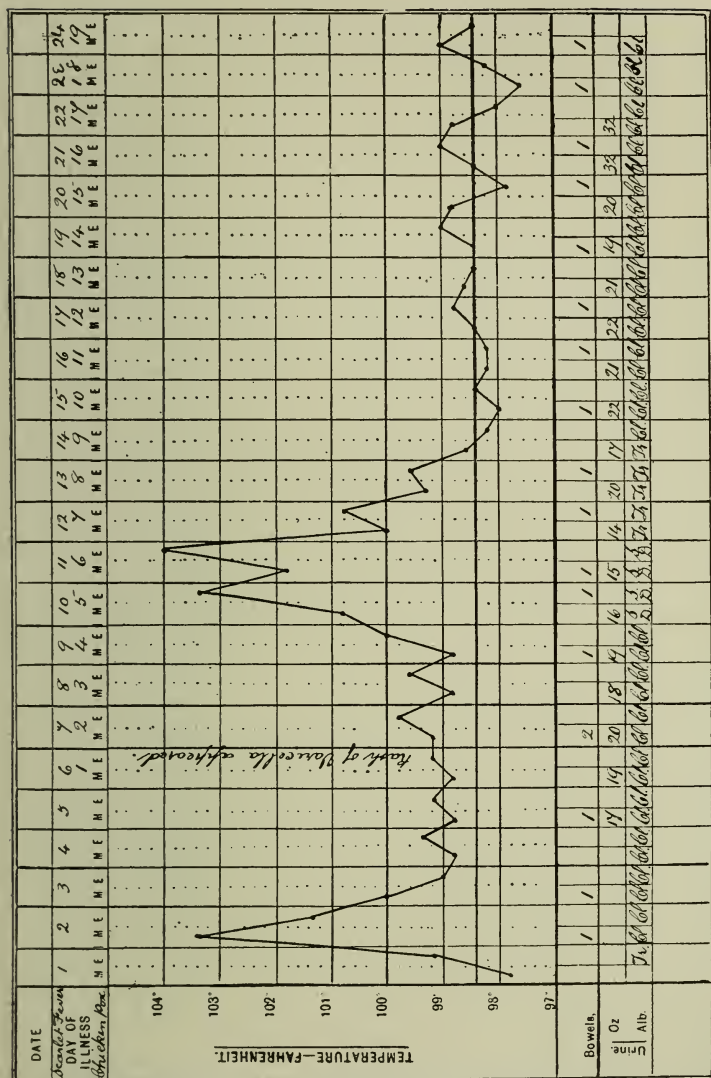
M. C., aet. $2\frac{8}{12}$. The illness was said to have begun 3 days ago, but, on account of the diagnosis being doubtful, the child was placed in an isolation ward. On admission a faint blush was seen on the chest, and the urine contained a trace of albumen.

1st day. The child has been 10 days in hospital, and up till to-day has been quite well; no albumen found beyond a trace on day of admission. Became restless, and the temperature rose to 104° F. A well-marked scarlet rash seen on the chest. Throat congested; tongue clean.

6th day. Papules and vesicles of varicella seen to-day.

7th day. The eruption has become profuse, and has spread over the whole body.

11th day. The temperature last night was 103.4° F., and patient was very restless. The urine contained albumen this



SERIES II., CASE I.

morning, sufficient to form a small deposit. No blood present. Microscopic examination of the urinary sediment revealed the presence of epithelial casts and renal epithelial cells undergoing fatty degeneration.

13th day. Vesicles drying; large number of crusts. Urine contains a faint haze of albumen.

15th day. Urine clear.

51st day. Urine has remained clear. Dismissed; completely recovered.

CASE II. (*See Chart.*)

J. N., aet. 6. Admitted on the 5th day of illness. Well-marked punctate rash, generalised. Fauces congested and swollen; cervical glands normal. Tongue clean, red, moist. Temperature on admission, 102° F.; pulse, 144; respirations, 48. Urine normal.

22nd day. Patient was going on well till yesterday, when he had diarrhoea, and in the evening his temperature rose to 99·6° F.; this morning it was 102° F. The cervical glands are swollen. Face pale and puffy. Small deposit of albumen in the urine; blood also in tolerably large amount.

23rd day. Passed 37 oz. in last 24 hours; slept well; sick thrice yesterday; once after drinking and twice spontaneously.

24th day. Passed 15 oz. only; no complaint of pain; blood larger in amount; drinks well; was sick during the night.

25th day. Passed 18 oz. urine; skin moist.

26th day. Passed 15 oz.; deposit of albumen, $\frac{1}{2}$ column; less blood than previously; no vomiting; drinks well.

27th day. Passed only 7 oz.; larger amount of blood; vomited frequently this afternoon.

28th day. Slept fairly well during the night. Temperature this morning 102·3° F.; frequent inclination to be sick; passed 9 oz. urine.

29th day. Urine passed, 10 oz.; deposit of albumen, $\frac{1}{2}$ column. Blood moderate in amount.

30th day. Urine passed, 18 oz.; no vomiting; albumen same in amount; trace of blood.

Sphygmographic Tracings illustrating the occurrence of Cardiac Arrhythmia in relation

1894
Sept. 23
No. 1

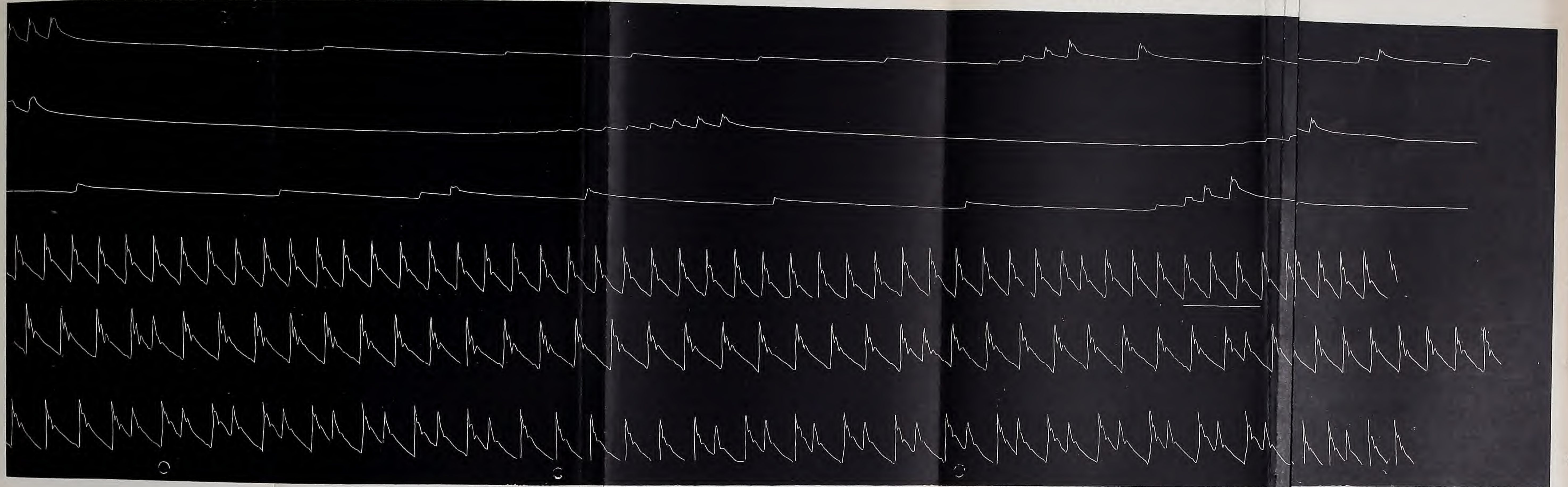
Sept. 23
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Sept. 24
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Sept. 26
No. 4

Sept. 27
No. 5

Sept. 27
No. 6





32nd day. Urine passed, 28 oz.; deposit of albumen, $\frac{1}{8}$ column.

37th day. Urine passed, 36 oz.; haze of albumen and trace of blood.

41st day. Urine passed, 49 oz.; haze of albumen; no blood.

45th day. Urine clear; 50 oz. passed.

47th day. Rash of varicella seen to-day; a few papules and vesicles have appeared on the chest and back.

48th day. Urine clear in morning; in evening contains a deposit of albumen; trace of blood.

49th day. 39 oz. urine passed; deposit of albumen; and blood in considerable amount.

52nd day. Faint haze of albumen; blood less.

53rd day. Haze of albumen, but no blood.

54th day. Urine free from albumen and blood.

78th day. Continued improvement.

83rd day. Allowed up. Dismissed; completely recovered.

CASE III.

L. D., aet. 6. Admitted on the 4th day of illness. Initial signs well marked, and little constitutional disturbance. Temperature on admission, 98.4° F.

15th day. Course has been mild. A rash consisting of numerous papules and vesicles has appeared; it is that of chicken pox. Evening temperature, 104° F.

23rd day. The eruption has been profuse and pustular; it is drying; the temperature is now about normal. The crusts are separating satisfactorily. A haze of albumen was detected for the first time.

26th day. Haze of albumen was present each day till to-day, when urine is clear. It remains clear afterwards. Dismissed; completely cured.

The principal points with which we are specially concerned here connected with the above cases may be presented in tabulated form thus:

No. of Case.	Albuminuria or Nephritis before onset of Varicella.	Day of illness of Scarlatina on which Varicella appeared.	Day of illness of Scarlatina on which Albuminuria or Nephritis occurred.	Day of illness of Varicella on which Albuminuria or Nephritis occurred.	Nature and Duration of Albuminuria or Nephritis after onset of Varicella (day of illness of Scarlatina).	Character of Varicellar Attack.	Result.
1	Neither	6th	11th	5th	Albuminuria, decided, 6th to 13th day	Eruption copious, Febrile disturbance severe	Recovered completely
2	Nephritis, 22nd to 45th day	47th	48th	2nd	Nephritis, returned, 48th to 54th day	Eruption moderately copious, Febrile disturbance severe	Recovered completely
3	Neither	15th	23rd	8th	Albuminuria, slight, 23rd to 26th day	Eruption copious, Febrile disturbance severe	Recovered completely

In Series II. it is chiefly noticeable that in all three cases the constitutional disturbance during the varicellar attack was severe, and that in two the eruption was profuse, and in the third moderately so. The interval also between the appearance of the varicellar eruption and the onset of albuminuria or nephritis varied from 1 to 7 days. Altogether, then, it may be said that while the albuminuria or nephritis more usually occurred early, viz., between the 1st and the 4th days, it occurred as late as the 11th day of the varicella.

Again, it is of importance to ascertain whether the occurrence of albuminuria or nephritis after the appearance of chicken pox had any special reference to particular periods in the course of the scarlatinal attack. For this purpose a small table has been made out which will enable to be readily seen what may be deduced from both series of cases on this point :

Course of Scarlet Fever divided into Weekly Periods.	Number of cases in which Albuminuria or Nephritis occurred after onset of Varicella.	Number of cases in which Varicella appeared.	Remarks.
1st week,	0	3	In one case the Varicella appeared at the beginning of the week, in the remaining two at the end of the week.
2nd "	4	2	
3rd "	2	2	
4th "	4	3	
5th "	0	0	
6th "	0	0	
7th "	3	3	
8th "	2	2	

From the above table it will be seen that the albuminuria and nephritis occurred practically in consonance with the incidence of the varicella, and was not specially related to any particular period of the scarlatinal illness.

The conclusion which was arrived at from a consideration of the first series of cases receives support from the facts supplied by the second series. Confirmation is likewise afforded in the fact that many records of cases of nephritis occurring as a complication of varicella by itself, as has been shown, have been published.

The result of our inquiry may be formulated thus :

I. That when the two diseases in question co-exist, the liability for albuminuria or nephritis to ensue, when neither complication has been present prior to the onset of varicella, is greater than in the case of scarlet fever alone.

II. That when such a complication has been present during the course of scarlet fever, and has been recovered from, it is more apt to recur in a mild or a severe form, with the onset of chicken pox.

III. That if the varicella appear during the albuminuria or nephritis, either complication is more liable to be aggravated.

IV. That the renal affection is, under these circumstances, more liable to assume a severe or even a grave form.

From the particulars furnished by both series of cases it will be seen that the severity of the varicellar attack bears a certain relationship to the occurrence of albuminuria and nephritis, as also the nature and distribution of the eruption. When it is profuse, of wide distribution, and the individual vesicles are large and show a tendency to run together, and to form flattened or umbilicated pustules, this circumstance is most conducive to the development of albuminuria or nephritis ; still, as will have been observed, either complication may develop when the eruption is moderately copious and the general constitutional disturbance inconsiderable.

The further question of how this tendency to impairment of the functions of the kidneys is to be explained is a pertinent one. It is, however, bound up with the larger one of the relation of micro-organisms to nephritis in the special infectious diseases, concerning which our knowledge is as yet limited. The present investigation, as bears on this point, was limited to the second series of cases, so that the answer to this question must necessarily be of an incomplete nature.

In the first place allusion must be made to the state of the kidneys in scarlet fever. I am not disposed to adopt the view that renal changes are invariably present in the course of scarlet fever, though that they are very frequent is, I think, shown by the investigations of Klein. He discovered the presence of definite glomerulo-nephritis in twenty-three

consecutive cases, fatal at periods varying from two days to seven weeks.

Caiger asserts that in at least one case, that of a patient who died early in the course of scarlet fever from a cause foreign to that disease, careful microscopical examination revealed no evidence of nephritis, either of the glomerular or interstitial form. And Bartels asserts that in many cases of scarlet fever the urine contains no renal epithelium. Wagner has found that in many cases of scarlet fever the kidneys were quite intact when submitted to examination. Certain changes, be they slight or severe, are no doubt to be found in a large number of cases, although there may be only a slight form of albuminuria present during life. It is, then, with the minute structural changes in the organs in question that we are chiefly concerned, and with what we can learn regarding the processes which have been going on in these same organs during life.

The earliest changes that occur in the kidneys during the course of scarlet fever are vascular. The vascular vaso-motor apparatus is, as we can understand, peculiarly exposed to the direct action of any irritant which has obtained access to the general circulation, and in particular to that part of the vascular system belonging to the excretory organs, and therefore, concerned in the elimination of certain waste products and deleterious substances from the blood. Those changes, then, that occur in the form characteristic of scarlet fever involve chiefly, on the one hand, the vascular structures referred to, and, on the other, the epithelial elements of the excretory apparatus, and more particularly those of the capsule and convoluted tubules. And while the latter usually occurs as a later condition, it may exist along with the alterations in the vascular structures, and either the one or the other may preponderate.

The diffuse nature of the inflammation, and the circumstance that the principal structures involved are those concerned in the separation and removal of effete products, or foreign substances, from the circulatory system, point to the cause as being some virus brought to the kidneys in the blood stream. Micro-organisms have been found in various structures

in those organs in scarlet fever. The late Professor Coats, in his *Manual of Pathology*, 1883, p. 709, mentions that in those cases colonies of bacteria are sometimes to be seen in the capillaries and uriniferous tubules of the kidneys, and that it is not improbable that we have here to do with the specific organisms of the fever. Pathogenic streptococci were likewise found by Lustgarten and Mannaberg, also by Turner, who, however, remarks that a considerable number of examinations were made with only a few positive results, most of the cases examined being of the septic type. As the result of his experiments, he found micro-organisms more frequently present in the glomerular than in the septic form.

Wyssokowitch declares that the kidneys in an intact condition do not excrete bacteria, but that the passage of such bacteria necessitates a local lesion in the renal parenchyma. His conclusion was founded on experiments apparently carried out with much care. And Fürbringer states that recent experiments, carried out according to the Gram method, prove that the kidney, in inflammation of infectious origin, can eliminate pathogenic germs, but that those germs can by no means be considered as primary agents of the disease. It is therefore fitting to inquire whether any other proximate cause is to be found which might give rise to the appearances and clinical signs mentioned. It has been shown that certain bacterial products or toxins circulating in the blood may produce lesions in various organs unassociated with the actual presence of bacteria. The secretory cells of various organs, and in particular the liver and kidneys, are specially liable to be affected. Cloudy swelling may result, and this may be followed by a fatty change, or by necrosis with granular degeneration. Hyaline changes in the walls of the arterioles are also mentioned as occurring. Capillary haemorrhages also are of frequent occurrence, partly from the vessel-walls being abnormally permeable, and partly from changes in the blood plasma. Many of these conditions are found in the kidneys in scarlet fever, as also the presence of blood and albumen in the urine during life. There is, therefore, a strong presumption that the renal inflammation is brought about by the action of soluble products of microbic origin.

It has been seen, then, that as the result of the action of agents which act deleteriously definite alterations of important structures concerned in secretion and excretion take place with comparative frequency in the course of scarlet fever. It will readily be understood, therefore, how a slight cause may suffice to induce nephritis if it has not previously existed, or to aggravate that condition if already present, or to light up the acute process afresh should resolution have been taking place. It has also been shown that nephritis may, and does, arise in the course of varicella by itself; hence it is natural to infer that when the structures referred to have been damaged in the course of scarlet fever, whether by an acute inflammatory process or by a sub-acute and less evident process, they are specially vulnerable, and the more so when the infectious disease superimposed shows a similar tendency to affect the kidneys, although to a much less extent than is the case in scarlet fever, measles, or diphtheria.

The aetiology of varicella, like that of scarlet fever, is obscure. Guttman, who made a series of bacteriological examinations of the contents of vesicles in patients suffering from varicella, has described three kinds of staphylococci, of which he obtained pure cultures. One was found to be pathogenic, and the other two non-pathogenic.

In the cases which we have just been considering, it has been shown that albuminuria or nephritis occurred in the majority of instances either immediately on the appearance of the eruption of chicken pox, or within a very few days after its first appearance, that is during the time the eruption was coming out in crops. I therefore sought to find out whether any micro-organism could be discovered in the vesicles in these cases which might be instrumental, directly or indirectly, in causing the renal mischief. Accordingly, a bacteriological examination of the contents of vesicles was made in each of the three cases belonging to Series II., in which I was aided by Dr. J. G. M'Coll, who was then Senior Resident Medical Officer at Kennedy Street Hospital. Cultures were made from the contents of mature and, as far as possible, somewhat large vesicles, and on the day on which the albuminuria or nephritis appeared, that is to say on the first, second, and fifth days

respectively of the varicella, also between the last mentioned day and the eighth day.

The result was briefly as follows :

Case 1.—(a) A staphylococcus which gave rise to a porcelain-like growth on gelatine; the gelatine was not liquefied. It responded to Gram's method of staining, and in its behaviour with respect to the various media employed it resembled the staphylococcus cereus albus; but when examined in stained film it was seen to differ from that organism, and in size and mode of grouping to resemble staphylococcus pyogenes albus. It could not be identified with any of the recognised forms of pyogenic cocci.

(b) *Sarcina lutea*.

Case 2.—Two serum agar tubes inoculated from vesicles on the second day remained sterile. On the sixth day staphylococcus pyogenes albus was obtained in pure culture.

Case 3.—A staphylococcus which, when inoculated on gelatine, gave rise to a greenish-yellow growth at the end of forty-eight hours; the gelatine was slowly liquefied. A staphylococcus described by Guttman as producing a lemon-yellow growth on gelatine presents a strong resemblance to it, but differs from it in not liquefying gelatine. When examined in stained film it was seen to resemble the staphylococcus pyogenes aureus in size and mode of grouping, but not in its behaviour with respect to the various media.

Permission was not sought to conduct inoculation experiments on animals. Still, it is highly probable that, with one exception, the micro-organisms found were non-pathogenic, and that they penetrated into the interior of the vesicles from the outer surface. Certain pathogenic microbes may, however, find their way into the interior of the vesicles from the surface of the skin, as has been shown by Garré as the result of culture experiments. Still, the fact that in seven of the total number of cases, namely, fifteen in which albuminuria or nephritis occurred on the first or second day after the appearance of the eruption, and that in eleven it occurred on or before the fourth day, and also that no micro-organism apparently of any pathological significance was found during that period, would seem to indicate that the determining cause of the renal complication

is not to be looked for in an actual contagium vivum, but rather would it seem to be due to the presence of certain soluble alkaloids. Especially is this so in view of the fact that many of the symptoms and signs in infectious diseases, such as certain rashes and the febrile condition, can be reproduced artificially by the injection of toxines, and that an analogy exists in the action of certain drugs. Moreover, the very recent experiments of Lindemann lend striking confirmation to this view. He found that a powerful "nepholytic" action was exhibited when kidney substance of an animal of one species suffering from acute nephritis, finely pounded, was injected into an animal of another species, and the serum from the latter injected into an animal belonging to the former species. He also produced acute nephritis in dogs by administering chlorate of potassium. He then injected the serum of these dogs into others, with the result that albuminuria, suppression of urine, and uraemia were produced.

Proof has then been adduced that nephritis may arise independently of the direct action of micro-organisms, and there is a strong presumption that the nephritis and albuminuria arising in the course of scarlet fever, complicated by chicken pox, originates in this way.

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ABBREVIATIONS USED IN TEMPERATURE CHARTS.

Tr.=Trace ; refers to albumen or blood. Trace of albumen,—sufficient to produce a haze or cloud on boiling and adding dilute acetic acid. Trace of blood, or blood colouring matter,—when the urine responds slowly to the guaiac test.

S.D.=Small deposit of albumen, $\frac{1}{8}$ column and less.

L.D.=Large deposit of albumen, more than $\frac{1}{8}$ column.

Cl.=Clear, or free from albumen.

S.A.=Small amount, referring to blood ; urine smoky, and responds readily to guaiac test.

L.A.=Large amount, referring to blood ; urine bright or deep red in colour.

UGHT TUBERCULAR DISEASES TO BE TREATED IN THE SURGICAL WARDS OF A CITY GENERAL HOSPITAL? A STATISTICAL STUDY.

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It is hardly possible for any, except those in professional and personal charge of a surgical ward in one of our large general hospitals, to properly appreciate and weigh with serviceable intent the many bearings which tubercular diseases possess, when considered in relation to the work as a whole, carried out on the surgical side of the institution. The annual reports of our infirmaries are not sufficiently exact, nor so uniform in their classification of the diseases treated, as to render it possible to make use of them for any strictly professional purposes. Even supposing an attempt were made to classify under one head all diseases described as tubercular, it is doubtful whether these reports would then be sufficiently reliable for technical statistical purposes. Unless special note were taken, and every endeavour made to ascertain whether each case admitted into hospital was or was not one which should be considered tubercular, the number of cases certified as such would probably be considerably under the actual number treated.

So far as our Glasgow Infirmaries are concerned, the annual reports attempt no such classification, and we, therefore, find that tubercular diseases are distributed under many different heads, and not only so, but in several classes where certain diseases may or may not be tubercular—as, for instance, mastoid disease—there is no means of knowing to what cause the affection should be attributed.

For these various reasons, therefore, the relation which cases of tubercular disease bear to other classes of disease treated in a surgical ward can only be ascertained with any approach to accuracy, and utilised for any practical and serviceable purposes, through the personal observation and record of the surgeon in charge. With him alone rests frequently the decision as to whether the disease in any particular instance owes its origin to tubercular infection or not. Unless, therefore, this differentiation be made in every case, it becomes impossible by any other means to ascertain accurately the relative numerical proportion of cases of tubercular disease admitted or to arrive at any other conclusion where comparisons are needed.

With the object of obtaining some sort of an estimate regarding the number, nature, and gravity of the cases of tuberculosis admitted into my wards at the Victoria Infirmary, I have since the opening of the institution kept an accurate record of every case. The age and sex of the patient have been noted, the number of days of residence in the ward, the region affected, the treatment adopted, the result, and such other incidental facts that seemed of technical interest. I had not contemplated making an immediate use of these records, but, as the editors of the *Glasgow Hospital Reports* have again kindly asked me to contribute, it occurred to me that such general information as it seemed possible to derive from a consideration of these personal records might prove of a kind peculiarly suited for such a periodical as our "Annual Reports."

Although the Victoria Infirmary has been opened for about twelve years, I have made use only of the last five for the statistics given below. It is within this latter period that my two male and female wards have been in full working order.

I may indicate, in the first place, that I only intend drawing upon my records for such information as will serve the particular purposes I have in view. There are naturally many other facts capable of deduction from the same sources. These, however, will be kept for possible future use. What I want to consider and discuss in the present paper may be thus briefly indicated:

- (1) The proportion which cases of tubercular disease bear to other diseases treated in a general surgical ward.

- (2) A limited classification of diseases dependent upon tubercular infection, with special reference to the parts or regions most frequently affected.
- (3) The period of residence in hospital of cases of spinal disease and hip joint disease treated more or less to a conclusion.
- (4) The ages at which tubercular diseases are most frequently met with.

And then in conclusion I shall make such comments and proposals as I think a consideration of the above points reasonably warrants.

I may state that the accommodation afforded for the cases to be considered comprises two large wards—a male and a female—each having eighteen beds and three cots; two special rooms, one containing three and the other four beds; making a total of forty-nine beds.

Discussing the points in order, as above indicated, the following table (A) shows the relative proportion of tubercular and non-tubercular cases admitted within the five years:

TABLE A.¹

Percentage of Cases of Tuberculosis admitted within the five years, 1896-1900.

Date.	Tubercular.	Non-Tubercular.	Total Admission of all Cases.	Percentage of Tubercular.
1896	107	290	397	26·9
1897	108	323	431	25·0
1898	115	324	439	26·1
1899	108	359	467	21·2
1900	114	364	478	23·8
Totals	552	1660	2212	—
Average Admissions per year	110·4	332	442·4	24·9

¹It should be stated in connection with the table that the numbers in columns 2, 3, and 4 have reference to "admissions" and not to the actual number of separate individual cases; that is to say, among both classes, whether

Taken over the whole period of five years, it will be seen that the proportion of tubercular to non-tubercular cases admitted is practically one-fourth.

The next table (B) is intended to give some idea of the nature of the cases and the numbers of such cases admitted.

TABLE B.

Shows relative frequency with which certain regions of the body are infected with Tuberculosis, and the number admitted each year from 1896-1900.

Date.	Spine.	Hip Joint.	Knee.	Ankle.	Shoul-der.	Elbow.	Wrist.	Cervi-cal Glands.	Others.	TOTAL.
1896	14	8	6	4	1	6	1	22	45	107
1897	8	18	6	5	1	6	—	32	32	108
1898	10	12	8	7	2	4	1	22	49	115
1899	10	13	10	4	2	7	1	21	40	108
1900	11	17	4	1	3	5	1	15	57	114
Totals	53	68	34	22	9	28	4	112	223	552

A glance at the above table shows that so far as the classification has been carried out, cases of infected cervical glands are among the commonest; and that among joint cases, those of the hip and spine take a somewhat long lead of the others. The knee and the elbow stand about equal in frequency; followed, however, somewhat closely by the ankle. The wrist and the shoulder joints stand very markedly the lowest. Diseases of the hip joint are, I believe, by common consent, allowed to be those most frequently met with. In four out of the five years in the above table, the admissions for this disease were in excess of any other, and only in 1896 were they surpassed by diseases of the spine. Taking the two diseases together, spine and hip, they far and away numerically

tubercular or non-tubercular, no account has been taken of re-admissions, in the sense that the same cases admitted a second or third time are reckoned as two or three cases. It was considered too difficult to take this item into consideration, and while it naturally reduces the actual number of individual cases of disease, the fact that both classes are treated in the same way renders the percentage comparisons practically correct.

exceed the sum total of all the other joint cases. In an interesting and instructive paper¹ read before the Glasgow Medico-Chirurgical Society on December 7th, 1900, Dr. Grant Andrew shows somewhat more accurately than is indicated above the true proportion of hip joint cases "admitted" to individual cases of disease. Thus 92 "admissions" represented 70 separate cases. And again, in calculating the average length of time of residence, Dr. Andrew, by considering the "re-admissions," finds the period to be 150 days, as against my average of 128·5 days shown in Table C. These differences, however, do not constitute inaccuracies, they simply exhibit an aspect of the question viewed from two different stand-points.

TABLE C.

Shows average period of residence in Hospital of Hip and Spine Cases subjected to more or less radical treatment.

Date.	SPINAL CASES.			HIP CASES.		
	No. of Cases.	Case of longest residence in days.	Average No. of days in Infirmary.	No. of Cases.	Case of longest residence in days.	Average No. of days in Infirmary.
1896	6	340	129·1	7	269	147·
1897	3	70	64·6	16	359	166·6
1898	7	169	78·8	8	253	108·2
1899	5	128	88·2	11	312	115·5
1900	7	280	125·9	16	363	101·3
Totals	28	—	101·5	58	—	128·5

The next table (C) is drawn up to show the average length of residence of spinal and hip cases in hospital. The average is calculated only on those cases that have been treated to anything like a conclusion. Many incipient cases of both spine and hip joint disease are taken in only for a few days to have a jacket or a Thomas's splint applied. These, while they

¹ *Tubercular Disease of the Hip Joint: A Critical Examination of 70 Cases treated in Mr. Maynard's Wards of the Victoria Infirmary, Glasgow.* By J. Grant Andrew, M.B., Assistant Surgeon to the Infirmary. Glasgow: Alex. MacDougall, 1900.

are included in the total number of admissions, have not been considered in taking the average, because some of them come in again for more radical treatment, and as such are subjected to efforts directed to effect a complete cure, thus rendering them proper cases to be included in the "average" list.

An examination of the above table shows that so far as the length of residence of any one case of either disease is concerned, there is practically no difference; but considered collectively, hip cases, as a rule, are in the infirmary for a longer period than spinal.

In the next table (D) I have sought to ascertain the ages at which tubercular disease most frequently shows itself; and for this purpose I have made two classifications, one, of patients under and patients over 40 years of age; and the other of quinquennial periods up to 20. The distinction of sex has not been regarded, nor the relative frequency with which certain parts or regions are affected at different periods of life. It may, however, be said that with children at least, sex appears to have little or no influence on one side or the other; and that in the matter of regional disease the joints and bones are the parts most frequently involved in the tuberculosis of early years.

TABLE D.

Shows relative frequency of Tuberculosis at different ages.

Date.	1 to 5 Years.	5 to 10 Years.	10 to 15 Years.	15 to 20 Years.	Total under 20 Years.	Total over 20 Years.	Total of all cases.
1896	24	26	12	17	79	28	107
1897	25	28	22	14	89	19	108
1898	29	27	19	13	88	27	115
1899	23	29	17	11	80	28	108
1900	13	30	19	9	71	43	114
Totals	114	140	89	64	407	145	552

It will be seen from the above table that there is a slighter increase of frequency of the disease between the ages of from 5 to

10 years than between the first and fifth year, but taken together the first decade of life shows a very much greater prevalency than the second decade, or indeed any subsequent period of life. Further, if we consider the total number of cases of the disease which occur in patients under 20 years of age, they are seen to nearly triple the entire collection of cases occurring at any period over that age.

Before passing to the conclusions I wish to draw, and the proposals I wish to make, I should like to offer a few remarks upon the subject of operative treatment. If twenty years of hospital work in the treatment of this disease in the surgical wards of a general hospital may be considered a sufficiently reasonable period to base an opinion upon the relative value of operative intervention, I must own to a resultant feeling of disappointment. Tuberculosis seems to be essentially—at least among the lower classes—a disease which finds its initiation and most fruitful source of development in ill nourished and badly cared-for children. The higher the social scale is mounted the more we find that the “predisposing” element plays the most prominent part. This same element, of course, exercises its influences upon the poorer classes, even to an exaggerated extent; and whatever else may enter into the causation of the disease, or contribute towards its manifestation, it is certain that impoverishment of the system by deficient and bad feeding, with the addition of an insanitary environment, constitute not the least important factors of the many agencies at work. In the few remarks just given expression to are to be found, I believe, the best indications for the most appropriate and successful treatment to be adopted, and also the reason why operations so frequently fail in giving us the good results we otherwise both look for but too often fail to obtain.

A couple of years ago I wrote an article for the *International Clinics*¹ on “Surgery in relation to Tubercular Diseases.” One of the contentions therein expressed was that we should consider tuberculosis in the same light that we regard carcinoma: that just as we deemed it imperative to remove most thoroughly and freely all parts involved in the malignant growth, so we should extirpate as completely all tuberculously infected areas.

¹ Vol. i., 9th series, April 1899, p. 179.

There is no doubt much truth in this, but it is subject to one very cogent qualification, and that is, that while in carcinoma, as far as we yet know, operation is the only means of effecting a cure, it is by no means so in the case of tuberculosis. Indeed herein lies, I believe, a distinction so great that this divergence of likeness renders it all but inappropriate to discuss to any further extent the question of similarity.

The most potent factor in the retardation of the development and progress of tuberculosis is in a very large number of cases to be obtained, not through the intervention of the surgeon's knife, but by the natural resistant powers of the tissues. If these can be raised to the normal standard they will of themselves repel the offensive onslaught of the tubercle bacilli and effect the necessary repair. The enunciation of these facts may appear somewhat trite, but although sufficiently well known they are introduced here more particularly with the object of enforcing and making clearer the conclusions I wish to draw from the tables given above.

I will now consider these conclusions, and, in the first place, briefly state them thus:

1. Tuberculous cases taken as a whole are not suitable for the surgical wards of a general city hospital.

2. All tuberculous cases should be dealt with outside the confines of our large towns, in such places as afford the best opportunities of carrying out the open-air method of treatment.

3. If it is only found possible to carry out the elimination to a partial extent, then hip and spinal cases should be the selected classes for separate treatment of the open-air kind.

In considering the first of these three conclusions, it will not be out of place to give some general idea of the amount of tuberculosis that exists in our city, as judged by the number of cases admitted into the surgical wards *alone* of our three infirmaries—the Royal, Western, and Victoria—and the Sick Children's Hospital. The total number of surgical beds used for general surgical purposes in these four institutions is 622. Referring to Table A it will be seen that out of the average admission per annum of 442·4 cases into my wards, containing 49 beds, there was an average annual admission of 110·4 tuberculous cases. Assuming then, as I think may

reasonably be done, that the admission into the other surgical beds of the above institutions is at the same ratio, we have for the total number of 622 beds an average annual admission of 1401.4 cases of tuberculous disease. This, it may be owned, reveals with approximate accuracy the excessive amount of tuberculous disease that exists in our midst; and yet it is only a fraction of the total number of sufferers, for the present statistics are limited to those cases that find their way into the surgical wards. There are the medical cases in the medical wards; the dispensary cases; those in other institutions like the Broomhill Home and the East Park Cottage Home; and that vast collection which inhabit the slums and densely populated districts of the city. The contemplation of this scourge as thus numerically presented does indeed afford material for the gravest reflection, and shows itself a subject worthy of the most earnest regard and consideration of those whose efforts are being directed rather towards the prevention of the disease than its cure.

It is, however, with the latter of these two considerations that the present remarks have to do; and it is in the admission of the large proportion of cases into the surgical wards that, I venture to think, a mistake is made both in regard to the best means being adopted for the particular class itself, and to the best interests of the many other cases of a different kind which, while quite suitable for admission, are kept waiting indefinitely owing to the overcrowded condition of our wards.

I have already argued that what most of these cases require is not immediate surgical treatment, but plenty of fresh air and good food. I am convinced that there is as much reason for subjecting these cases to the open-air method of treatment as there is for the more specifically considered lung cases. If it is contended that the benefit of this method of treatment in phthisis is due to the direct action of fresh air upon the tubercular lesion in the lung, then except we as completely expose other infected regions a comparison can hardly be drawn. But if, on the other hand, it is in reality the free and perfect oxygenation of the blood indirectly acting upon the infected pulmonary tissue, then this same revivifying influence must be felt by

every other tissue in the body equally and with a similarly beneficial result. Assuming, then, this to be the proper treatment, the surgical wards of our city hospitals are not the best places for receiving this class of cases. And in order that the necessities of the situation may be properly met, it is essential that institutions properly built and suitably equipped should be erected in the freely open and healthy parts of the country.

As regards other classes of cases, which, owing to the great pressure placed upon our wards through the admission of so many tubercular cases, there is, I think, a distinct injustice being done. While practically little or no advance has been made in the surgical treatment of a very large proportion of tubercular cases, immense progress has taken place in other diseases. Thus an enormous field of successful work has been opened up in the domain of abdominal disease, to mention only one department. To keep out cases from being admitted for which immediate and permanent good could be done is, in all truth, submitting our infirmaries to an unintentional and unnecessary abuse. Briefly, then, I consider my first conclusion sufficiently substantiated by these two facts:—First, that tubercular cases could be treated better elsewhere than in our city infirmaries; and, second, that by such elimination or exclusion more suitable and more deserving cases could be admitted.

My second conclusion, that tubercular cases could be better treated in free and open spaces outside our large cities, is to some extent supported by what has already been stated in discussing the first conclusion. But there are other facts besides those above given.

If fresh air is such a prime essential in building up and restoring the natural resistant forces of the tissues, it can only be obtained by locating these cases in areas where the requisite amount of space can be obtained and where there is an absence of those contaminating influences so inseparably connected with all buildings in or in the immediate neighbourhood of large towns. Further, the buildings required are of a totally different kind to those deemed necessary for the treatment of most other classes of cases. Thus they must be constructed on the principle of allowing as much of the open air as possible to find

its way into the sleeping apartments; and, by spreading the buildings outwards rather than upwards, of granting the utmost facility for the conveyance of patients into the open grounds whenever the weather permits.

Table D adds further support to the conclusion contended for. The large majority of cases are under 20 years of age. Out of a total of 552 cases 407 were patients under 20, and of these most were attacked during the first decade of life. Now, it is particularly at this actively growing period of life that the tissues are most susceptible to the beneficial influences of a good environment. A child feeds on fresh air and sunlight almost as greedily as it does on food, and the cheering and stimulating sights and sounds of nature are not without their good effects in restoring the undermined and enfeebled constitution of these usually sickly young patients to a condition of health and happiness. Operation also, when found necessary, will be executed, if not under any better conditions of equipment than at present, at least with every prospect of the patients making a more speedy and satisfactory recovery. The same good influences are at work for the better and more rapid healing of the wounds as for the tissues, which are naturally struggling to check the progress of the disease.

Supposing, then, that only cases under 20 years of age were treated in country institutions, the elimination of this class alone from our city infirmaries would have a very great relieving effect upon our wards, and remove a considerable amount of the injustice which, as already indicated, is involved in the compulsory exclusion of so many cases suited for admission, and better suited for treatment.

But this consideration of the selection of cases leads me to my third conclusion. Whatever divergence of opinion may exist with regard to tubercular cases considered collectively, there is little doubt in the unanimity with which most surgeons are inclined to regard the more limited class of hip and spinal cases. That these may with advantage be treated as a class quite distinct from all other cases of tuberculosis is sufficiently attested by the fact that one special institution, at least, has already been erected for the exclusive treatment of hip disease.

A reference to Table B shows how numerous these cases are, and how they numerically exceed any other individual class. Table C shows the relative period occupied by those cases when treated to a conclusion; or rather it should be said to an attempted conclusion, for even after so prolonged a residence many still leave the hospital with discharging sinuses, or some other conditions which indicate that the disease still lingers. These two considerations alone stamp this particular class of cases as the greatest offenders in the matter of prolonged and exclusive occupation of beds.

But there are additional reasons which make it specially advisable that hip and spinal cases should be treated in an institution of the kind here advocated. Operation is by no means the only and best treatment to be adopted in very many of these cases. Prolonged rest with the application of certain mechanical appliances is frequently the only requisite treatment, and for these patients to lie for months in a ward, requiring no surgical skill and the very simplest of nursing, seems an almost unnecessary waste and even abuse of the skilled resources maintained at such great expense in our general hospitals.

These particular cases are pre-eminently suited for treatment by the open-air method, and ought to be relegated to special institutions not equipped to the high and expensive state of perfection of our city infirmaries, but so organised and arranged that while fitted for such surgical treatment as may from time to time be necessary, they can have all the simple and comparatively inexpensive attention required for an open-air life.

If still another plea were needed in favour of the exclusive treatment of this class of cases it would be that—so far as hip disease more particularly is concerned—it is children that are most frequently the patients. They, as already shown, are of all the most susceptible to the beneficial influences of open-air treatment. Dr. Andrew, in the paper already referred to, has entered a very strong plea for the treatment of hip joint disease in separate institutions. The object of his remarks were almost entirely directed to the attainment of such an end, and the facts which he has been able to obtain

and utilise for the purpose in view will hardly fail to convince those who will take the trouble to consider seriously the subject of the advisability of such a line of action. On this question of age which I have just alluded to, Dr. Andrew states: "Taking all cases, the average age on admission was 11. More than half of the cases, however, were in children under the age of 10; taking these alone the average age was 6."

Many more reasons than those above given might be introduced in furtherance of the objects in view. Enough, however, I trust, has been said in support of the conclusions put forward to render them both acceptable and worthy of being acted upon in the interest of those for whom they are specially intended.

THREE CASES OF NERVOUS DISEASE: (1) CEREBROSPINAL MENINGITIS; (2) FRIEDREICH'S HEREDITARY ATAXY; (3) MARIE'S HEREDITARY CEREBELLAR ATAXY.

By T. K. MONRO, M.A., M.D.,

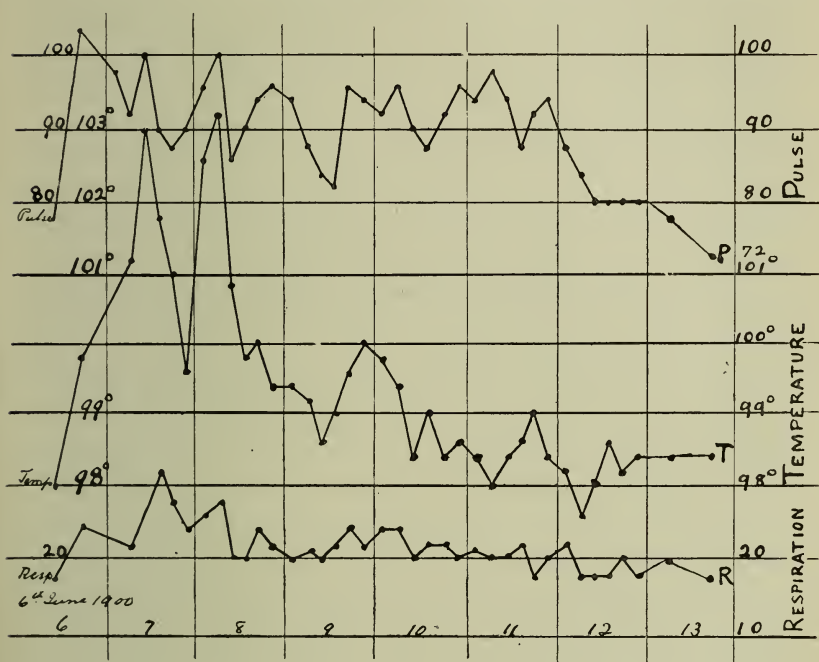
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(1) CEREBROSPINAL MENINGITIS.

MAGGIE C., aet. 14, was admitted to the Royal Infirmary on June 6th, 1900, on account of pain in the head and vomiting. She had been seized on the evening of the 3rd with shivering and giddiness, and an hour or two later with severe headache and vomiting, which persisted, with short intermissions, for about twelve hours. A doctor who was then summoned administered a powder, with the result that patient remained in a state of stupor for nine or ten hours. From the end of that period until her admission, she was very restless, and only conscious at times. She would start up and grind her teeth, and there was twitching of the limbs. The bowels were confined, and the urine was high coloured. No cause could be suggested for her illness, but it is perhaps worthy of note that in the middle of the day on which she took ill, she took onions, an article which, she said, never agreed with her, though she felt no inconvenience until nine or ten at night. Her previous history was a good one: she had measles in infancy, but never scarlet fever, or rheumatism, or any nervous ailment. The family history was excellent.

7th June.—Patient is at present to all appearance completely sensible, but is very restless, moving her trunk, tossing her limbs, and working her lower jaw almost constantly. The

face is much flushed, and the tongue dry and somewhat foul. Her principal complaint at present is of the bad taste in her mouth. Unless when questioned, she does not complain of pain, but she seems to have some about the back of the head or neck and in the left flank, and there is in addition tenderness on pressure in the left ovarian region and in the epigastrium. Nothing abnormal is detected on examination of the heart or lungs. There is an abundant eruption of herpetic vesicles around the right margin of the mouth: this appeared



on the morning of the day of admission. In the sitting posture, the head is thrown back, and patient is unable to move the chin downwards towards the sternum. When the head is passively depressed, patient complains of pain in front of the neck. The difficulty here is not due to any obvious spasm of the muscles of the neck. Patient generally lies with the knees and hips flexed, but it is possible for her to lie with these joints completely extended. Whenever she sits up,

however, flexion sets in, and when attempts are made to extend the limbs passively, patient either complains of pain or falls backwards. The abdomen is somewhat, though not extremely, retracted, and it appears that the bowels have not been moved since the 2nd inst. No rash is observed on the skin. The pupils are medium-sized or rather small, and contract actively to light. There is no strabismus and no detectable palsy of the face or tongue. It is noted that part of the right border of the tongue as well as the mucous surface of the upper and lower lips is involved in the herpes. A rough testing of hearing with the watch points to a considerable loss in each ear. There is no optic neuritis. There is distinct nystagmus when the eyes are rotated far to the left, and slightly on rotation upwards. There is none on rotation to the right. There is some tenderness over the spine immediately below the occipital bone, but not at any other point. The temperature rose from 98° on admission to 101.2° this morning, the corresponding pulse rates being 78 and 99, and respirations 19 and 22. Urine: specific gravity 1024, reaction acid, colour amber; flocculent sediment; slight trace of albumen; no sugar or blood.

8th June.—Giddiness is still present, though not so severe as at the onset. There is no intolerance of light or sound. There has been no delirium since admission. Patient slept during most of last evening and the early part of the night, getting perhaps in all seven hours' sleep, but since then she has been restless and uneasy and suffering much from pain all over the head, but specially about the occiput and back of the neck. There is great tenderness over various parts of the back, including two or three of the upper cervical spines and several of the lumbar spines. The tenderness is perhaps still greater at a little distance from the spine, and especially on the right side. It is also present in the suprascapular region on each side and in the lateral regions, and very specially in the right lumbar region external to the erector spinae. There is spontaneous pain also, especially in the lumbar region of the back. The pain is aggravated by movement, and may even be aggravated in the head by movement of the spine. In the front of the trunk there is tenderness to-day in the right ovarian region and

epigastrium. Patient speaks of spontaneous pain in the hips also. There is tenderness in the knees, but these joints can be passively moved without pain. The ankles are free from pain. The knee jerks are present. In the plantar reflex there is extension of the left great toe and also of the right (yesterday there was flexion of the right and extension of the left). The abdominal reflexes are deficient, but their investigation is interfered with by what appears to be slight hyperaesthesia of the skin of the abdomen, although, speaking generally, there is little or no hyperaesthesia of the skin. The eruption of herpes has been already alluded to. In addition, a certain amount of livid erythema was noted yesterday over the extensor surface of the left elbow. A similar appearance is present to-day over the right elbow as well, and to a certain extent over the extensor aspect of the knees. A similar condition is better seen to-day on the dorsal aspect of the right forearm at its lower part. This extends on to the back of the wrist, and is present in a more patchy form on the back of the forearm and hand, and is also faintly recognisable on the back of the left wrist. Patient lies preferably on the right side, with the hips flexed almost to a right angle and the knees flexed to about 135° . An effort to sit up this morning revealed continued retraction of the head, and almost immediately induced severe pain over the back and in the region of the stomach, which compelled patient to lie down at once. There is no noticeable rigidity of the limbs, and there is no discoverable palsy. There is no trismus. There have been no convulsions, either general or local. About 2 a.m. to-day, patient suddenly wakened out of sleep with a cry which seems to have been due to a sudden attack of pain which has been more severe in the head than elsewhere. The bowels were moved yesterday by enema, and have since then moved spontaneously. The bladder seems to be somewhat irritable, as patient makes more frequent attempts at micturition than are necessary. The herpes which is present on the right side of the face has not been associated with subjective sensations. Apart from the herpes no trophic lesion is detected in the area of distribution of the fifth nerve, but a slight conjunctivitis is indicated by a small quantity of pus at each inner canthus. There is no

enlargement of liver or spleen. Tache cérébrale, if present, is not very persistent. There is no hiccough or sighing respiration. The tongue, though still much furred, is moist and considerably improved as compared with yesterday, when patient was complaining of a bad taste. The parts on which patient rests are reddened in either a diffuse or a papular manner, and this is doubtless largely contributed to by her very restless condition. There is nothing, however, in the way of actual necrosis of tissue. There have been no mental symptoms since admission.

Patient states spontaneously that her throat has been sore for several days; it troubles her not so much in swallowing as in speaking.

9th June.—Patient is a good deal easier this morning, and is now able to sit up for a time. She is still unable to lower the chin to the sternum, and it is now ascertained that this disability is associated with spasm of the complexi muscles. The splenii, sterno-mastoidei, and trapezii are free from spasm, but there is much tenderness over the right sterno-mastoid and supra-spinous regions. Tenderness has passed away from the upper cervical spines, but is still present lower down. The only present seat of spontaneous deep pain is the knees. Constipation has now passed away.

11th June.—Great improvement in symptoms accompanied the fall in temperature, and, as far as the subjective aspect of the case is concerned, patient is now perfectly well. She is able to carry out normal movements without discomfort. The herpes on the face and tongue is rapidly healing. Some of the other cutaneous lesions are still recognisable, and in particular livid erythema on the right upper limb, red papules over the left olecranon and on each knee, and a vesicle below the right external malleolus.

No abnormality was ever detected in connection with heart or lungs.

After patient recovered, her hearing was tested more carefully than was possible in the height of the fever, and was found to be very defective. Thus, as tested with a watch, R. $\frac{1}{2}$, L. $\frac{1}{4}$. Rinne's experiment gave a positive result, but the excess of air conduction over bone conduction was

diminished. The membranes were found to be greatly retracted. Nevertheless patient asserted that her hearing was perfect before the illness which has just been described. She never had any discharge from the ears. The presumption is therefore that there was an old-standing dry affection of the middle ear, and that the present disease caused further impairment of hearing by damaging the internal ear.

Treatment in the febrile stage of the disease was by means of laxative enemata, two grains of calomel every eight hours, cold applications to the head and spine, and rest on either side or in the prone position instead of on the back. Later on, salicylate of sodium was given in combination with perchloride of iron.

The symptomatology in this case seems sufficiently characteristic. The headache, vomiting, delirium, retraction of the head, Kernig's sign, constipation, spinal tenderness, pain in the back aggravated by movement, etc., all point to cerebro-spinal meningitis; and the sudden onset with shivering and giddiness, the fever, the herpes, erythema, and other cutaneous lesions, the pains in the joints, and deafness complete the resemblance to the epidemic disease, of which this may be regarded as a sporadic example. Bacteriology might have settled the diagnosis beyond question, but an attempt at lumbar puncture was unsuccessful, one of the difficulties being the severe burning sensation experienced by the patient on the application of a freezing agent to the skin of the back.

(2) FRIEDREICH'S HEREDITARY ATAXY.

Jane H., aet. 25; no occupation. Admitted to the Royal Infirmary, 2nd April, 1900, complaining of dizziness, with weakness and stiffness of the legs.

Past history and condition on admission.—Patient belongs to a healthy family, and had no serious illness until the present trouble set in at the age of 15, after a severe fright which she sustained through being chased by two men on a dark night. From that time she was unable to carry on her occupation as domestic servant. Her first symptoms are described as dizziness and light-headedness, and these were constantly present,

but were specially troublesome when she walked in the dark, thus causing her to stagger and sometimes to fall. The next symptom was occasional pain about the upper part of the sternum, gradually passing upward into the neck and giving rise to a sense of constriction of the throat; this only troubled her on physical or mental excitement, and was relieved by a drink of water or by loosening the neck of her dress. When aged about 20, she began to have slight shooting pains in the lower limbs from the knees downward, and especially in the ankles and feet; these pains, which are still felt at times, were of momentary duration, and did not occur oftener than once a day.

About the same time, she first suffered from a feeling, sometimes painful, as of a band round the body; this has become more severe of late, and is constantly present, though aggravated by excitement or exertion. It is sometimes associated with a sense of abdominal distension, which compels her to loosen her dress at the waist. The principal seat of this girdle-sensation is the epigastrium. The upper limit is about the level of the fourth costal cartilages, and the lower limit about midway between the xiphoid and the umbilicus.

Since the New-Year, patient has been subject to a severe pain, which occurs in paroxysms at the right lower costal margin, towards the back, and when this sets in patient ceases to be sensible of the abnormal sensation in the epigastrium. She states that the severe pain in the side comes on only when she is walking and happens to look to the ground; it is associated with dizziness so severe that she falls backwards. She has occasionally had pain in the stomach after food, but did not vomit at these times. The bowels have long been constipated. When she is going about on her feet she has an almost constant inclination to pass water, and occasionally, if she does not make haste to empty the bladder, urine will escape involuntarily, though not without her knowledge. There is no trouble of this kind when she is in bed, and there is no delay when she wishes to evacuate the bladder. Menstruation has always been regular.

There is well-marked slowness of speech, of which patient is quite aware, though she has no idea when it commenced. At

times there may be noted a tendency to separate the syllables of words, but the main defect is simply slowness of utterance.

Though patient complains of both weakness and stiffness in the *legs*, no definite weakness can be detected as she lies in bed, and no tonic or clonic spasm can be recognised in these limbs; but when she walks there is a perceptible tendency to drag the feet, and especially the right foot. The freeness of movement in the toes and the slight disorder noticed in the manner of putting down the feet suggest that the difficulty in walking is due to incoordination more than to weakness, and this is borne out by the fact that she cannot keep her balance when she tries to stand with her feet close together,—a disability which is rather increased when she shuts her eyes. She can stand however, with the feet apart, even if the eyes are shut. In walking, as in standing, the ataxy is distinct but not extreme. There is no club-foot. The right knee jerk is normal or slightly sluggish; the left is diminished. The plantar reflex is represented by flexion of the toes on the right side, and is absent or represented by extension of the great toe on the left side. The sense of posture in the legs is good, but the common muscular sense appears to be much diminished in the muscles of the calf. The ground feels normal to the feet. When the various other forms of sensation in the legs (touch, pain, temperature) are examined, it is not possible to detect any localised defect, but there is a general lack of acuteness, as shown, for instance, by the way in which patient may attribute the touch of the finger to a pin point, or may fail at one moment to recognise the piercing of the skin by a pin, while showing a little later that the sense of pain in the same part is well preserved. After a little practice, patient indicates when she is touched with almost no delay more than the normal. There is no history of perverted sensibility in the legs, such as a sensation of pins and needles. The legs when exposed readily show a livid marbling. There is no muscular wasting.

The only symptom referable to the *upper limbs* is occasional slight pain or stiffness at the right elbow and right metacarpophalangeal articulations, which began about a week ago. There is no localised palsy or wasting.

There is well-marked curvature of the *spine*, viz., kyphosis

in the dorsal region and lordosis lower down. In addition, there is slight skoliosis, with the convexity to the right at the level of the scapula. When she pushes with the left arm, a groove appears along the inner border of the left scapula chiefly at its lower part. No such groove is seen when the upper limbs are at rest by the side, and the effect of a strong pushing effort is not only to make the lower scapular angle more prominent, but also to tilt it slightly inwards (paresis of serratus magnus). There is no trace of any such weakness on the right side, and there is no evidence of weakness of the rhomboids. The power of the trunk muscles, as tested by efforts to raise the head and shoulders in the prone and supine positions, is regarded as normal. It is perhaps worthy of note that patient finds she can keep her balance much better in walking when her hands are placed over the front of her chest than when they are allowed to hang at her sides.

The abdominal and epigastric reflexes are well marked.

Cranial Nerves.—i. Smell is preserved.

ii. V.A. Right and Left each about $\frac{6}{36}$. This defect may perhaps be explained by the hypermetropia or hypermetropic astigmatism which is recognised by the ophthalmoscope. No hemianopia. Fundi normal. Examination of the visual fields shows only a slight restriction in the outer parts.

iii., iv., and vi. The functions of these nerves may be regarded as normal. The pupils are equal and somewhat large; each contracts directly and consensually to light; each contracts in convergence of the visual axes; and each dilates when the skin of the neck is stroked, either on the same or on the opposite side. Occasional slight jerks are seen when the eyeballs are moved laterally, but this scarcely amounts to nystagmus. There is no diplopia.

v. Motor function apparently normal, but difficult to investigate because of the difficulty in making patient understand instructions given. Common sensation in the region of this nerve normal. Taste is not very acute. Thus, while she recognises bitter and to some extent salt and acid solutions, she is scarcely able to recognise a sweet taste.

vii. There is a slight flattening of the left naso-labial furrow, but no other evidence of weakness of the muscles of

the face. The difference between the two sides is if anything less marked when patient tries to show her teeth.

viii. Hearing good in both ears.

ix., x., xi. There is no evidence, as judged by the condition of deglutition, respiration, vocalisation, etc., of any involvement of these nerves.

xii. Movements of the tongue normal.

There are no definite *mental* symptoms. The one feature in her case which suggests anything of the kind is her inability to comprehend some of the instructions that are given her in the course of examination. To this may perhaps be added the slowness of speech already described.

The only noteworthy feature in the chest is the presence of a V.S. murmur at the pulmonic area and to a less marked degree at the aortic area and manubrium sterni. Lungs normal. Temperature normal or subnormal. Urine contains a faint haze of albumen.

In the course of her residence, which extended over several months, patient's condition improved in some ways. Thus the slight pains in the right upper limb ceased, and the girdle-sensation also passed away. The attacks of pain in the right side became slight and infrequent, and any difference between the two sides of the face became inappreciable. The shooting pains in the legs were replaced by abnormal sensations in the feet and ankles, which at some times took the form of a sensation of pins and needles, and at other times of simple soreness. In the main, however, the symptoms referable to the nervous system persisted. The plantar reflex was for a time at least absent. The knee jerks were inconstant. The tendency was for both, but especially the left, to be deficient, even when reinforced. But the right was sometimes normal, and on one occasion seemed to be if anything exaggerated. The left knee jerk was sometimes slightly diminished, and at other times almost completely absent. There was no trophic lesion of skin or joints.

Examination by faradism and galvanism of different nerves and muscles of the lower limbs revealed no evidence of disease.

The haemic murmur became less marked, and an examination of the blood at this time gave the following results:—Fresh

blood microscopically examined presented normal appearances. Haemoglobin 70%. Red corpuscles 4,666,000, and white corpuscles 6400 per c.m. In a stained specimen, the four principal varieties of white corpuscles were found to be present in normal proportions.

The urine continued to show a slight haze of albumen.

The treatment was at first by arsenic with *nux vomica*. About six weeks later, this was replaced by phosphorus pill, for which, five weeks afterwards, Blaud's pill was substituted.

While the symptoms in this case are in the main sufficiently characteristic, one or two unusual features are present. The diagnosis of Friedreich's hereditary ataxia seems justified by the incoordination in the lower limbs with a comparatively slight degree of weakness, the late and trifling involvement of the upper limbs, the commencement at puberty, the slowly progressive course, the affection of speech, the spinal curvature, and it may even perhaps be added, the basic cardiac murmur. Among other evidences of spinal cord disease are the pains in the legs and trunk, the constipation, and the impaired control over the bladder. A very unusual feature, however, in this case is the preservation of the knee jerks, though it will be noted that there is deficiency on one side. Another departure from type is witnessed in the fact that this girl alone of her family suffers from the disease. A tendency to nystagmus is just recognisable. Other points which deserve attention, though not to be discussed here, are the different curves of the spine, the paresis of the left serratus magnus, the doubtful paresis of the left midface, the slight defect of taste and of tactile and painful sensation, the persistent slight albuminuria and the continuance of regular menstruation.

(3) MARIE'S HEREDITARY CEREBELLAR ATAXY.

Alexander S., aet. 15, was admitted to the Royal Infirmary on 12th April, 1900. Patient is the second child in a large family of children otherwise healthy, and born of healthy parents. He had measles in childhood, and thereafter enjoyed good health until aged about 12, when he had enteric fever. He never had scarlet fever. At about 13, it was noticed that

he became giddy after walking a short distance, and had to stop or hold on to some support. Patient seems to have been first conscious of his illness one day in school, when he was seized with shivering, which was followed by loss of feeling in the feet and legs. The disorder in the lower limbs gradually increased, so that he staggered when he walked, and sometimes fell. He was in the Victoria Infirmary for some weeks in 1899. He has had no pain or sickness, and there is little or no loss of control over the bladder and rectum. The bowels tend to be relaxed.

The following data were obtained after his admission to the Royal Infirmary. Patient's principal complaint is of weakness in the legs and feet, and indeed of weakness all over, but there is no localised paresis of muscles. There is no tonic or clonic spasm, and no tendency in walking to lift the feet unduly high. There is only a little tendency to drag the right foot. The difficulty with the legs is due to incoordination, which is such that he cannot stand with his feet together, and requires, when walking, to keep the feet far apart. The unsteadiness on standing is not much increased on closing the eyes. The knee jerks are greatly exaggerated, but no ankle clonus is obtained. The cremasteric, abdominal, and epigastric reflexes are diminished. The plantar reflexes are not obviously disordered, but are difficult to investigate on account of patient's sensitiveness to tickling.

The different forms of sensation in the lower limbs (touch, pain, temperature, posture, common muscular sense) are all normal, and there is no delay in the conduction of tactile impulses.

The *upper limbs* are unaffected.

The muscular weakness is most manifest in the trunk, being shown by the inability to sit upright for any length of time, but it can also be recognised in a slight degree in flexion of the ankles. The extensor muscles at the hip and knee retain great power, as is shown by the little assistance required to enable patient to step on to a bench. Both in sitting and on standing there is marked kyphosis, the head and shoulders being thrown far forwards, as if to counteract the tendency of the trunk to fall backwards. The extensor muscles of the

neck are powerful, and patient is able by an effort, when he is asked to do so, to make the back quite straight, at least when he is sitting in bed. He is quite unable, however, to maintain the sitting posture for any length of time, and tends to fall backwards and to the right. The power of raising the head from the bed, when the body is in the prone position, seems to be rather less than normal. There is no lateral curvature of the spine. There is no detectable atrophy or pseudo-hypertrophy of any set of muscles.

A certain amount of *intellectual defect* is present. Patient states that when he left school a year ago he was in the fifth standard. Whether this is correct or not, his arithmetic is now very rudimentary, and he fails to answer correctly such a question in multiplication as 7 times 5.

Another feature in his case is the defective power of attention, which is readily recognised in course of conversation with him. He is slow to answer questions, partly perhaps through lack of attention and partly through lethargy of mental processes. He is very emotional.

Although his *speech* is somewhat slow, there appears to be no defect in articulation as regards words or letters. There is no tremor of the tongue.

Cranial Nerves.—i. Smell appears to be acute, but it is difficult to investigate on account of the mental defect.

ii. Vision—Right, $\frac{5}{20}$; Left, also about $\frac{5}{20}$. O.E. normal.

iii., iv., and vi. There is no strabismus. Patient speaks of having once seen an object double, but no reliance can be put upon his statement. Nystagmus is present in well-marked degree on lateral movement, and to a less degree on upward movement of the eyeballs, but not spontaneously. Each pupil contracts directly and consensually to light. The pupils also contract in convergence. They are medium-sized and equal.

v. Taste in front of the tongue for sweet, salt, and bitter is preserved.

vii. Facial movements normal.

viii. Hearing good.

ix., x., xi. No symptoms referable to these nerves.

xii. Tongue normal.

Examination of the heart, lungs, spleen, liver, and abdomen generally yields normal results.

Urine normal as regards reaction, specific gravity, and absence of albumen.

The *bowels* still tend to be loose, the motions being watery. There is some defect of control over the sphincter ani, so that, although patient always gives notice, he has on one or two occasions been unable to retain the evacuation until attended to. There has been no such trouble with the bladder.

Temperature generally subnormal—never febrile.

In its more obtrusive features, such as the incoordination in the lower limbs, commencing in early life and slowly progressive in course, the muscular weakness and the nystagmus, this case is at first suggestive of Friedreich's disease. The great exaggeration of the knee jerks, however, together with the striking loss of power of the muscles of the trunk and the intellectual defect, seems to ally this case more closely with the special group which Marie called hereditary cerebellar ataxy, and which he regarded as depending upon some congenital defect in the cerebellum.

TRAUMATIC OCULAR PARALYSIS, WITH RECORDS OF THREE CASES.

By LESLIE BUCHANAN, M.D.,

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THE whole subject of ocular paralysis is one of so great interest to all branches of the medical profession, that no apology is required for bringing forward this most interesting section of it.

Leaving aside the large group of cases in which paralysis of ocular muscles is produced by inflammatory lesions of rheumatic or syphilitic origin, by vascular changes and by neoplasms of diverse characters, only those cases which are the direct result of injury to the head from the exterior will be considered.

Three cases will be briefly recorded to illustrate some of the various kinds of primary injury which cause such lesions, and also because they are of special interest in themselves in so far as in each only one of the group of muscles controlled by the third nerve is involved.

This type of case, then, can be divided conveniently into two classes, namely, (*a*) those in which there is evidence of direct injury to or near the orbit, whereby either the muscle or nerve affected may be injured, and (*b*) those in which there is no such evidence of direct orbital injury.

In the first variety the diagnosis is frequently easy. Some evidence of injury, such as ecchymosis or emphysema of eyelid or conjunctiva, irregularity of the bony walls, or pain of a localised character on pressure or on movement of the eyeball, usually remains for some time even if no skin or conjunctival wound be visible.

The injury to the nerve or the muscle may be either direct or indirect in nature. In the former case bruising or rupture may occur as a result of the penetration of a sharp or blunt instrument, whilst in the latter, owing to fracture of the orbital wall at some point probably posterior, a spicule of bone may be depressed and so give rise to injury.

The following is a case of the first class :

CASE I.

A man, A. M., aet. 27, was attending a snow plough when a chain snapped, and he received a sharp blow on the left lower eyelid at the inner part. The wound bled freely, but a local doctor who saw the man considered that it was quite a superficial injury. Soon, however, it was noticed that the patient could not move his eye completely and that the sight was defective. When the man came to the Eye Infirmary a week later, it was found that there was a small wound 4 mm. in length in the lower eyelid and discharging very slightly, that there was complete paralysis of the internal rectus muscle, and, lastly, that there was separation of the retina. The wound was probed and a foreign body detected about 36 mm. back in the orbit (this was in 1891, before Röntgen's discovery). Much difficulty was experienced in extracting this foreign body, but when this was accomplished it was found that it was half of a link of a chain made of quarter inch iron rod. This had been driven into the orbit with great force, and had been lying behind the eyeball. It was evident that in its passage backwards this mass had seriously injured the internal rectus muscle, whose power was never restored.

Such direct injuries frequently give rise to fixation of the globe by fibrous tissue formation, a condition known as fixed strabismus.

When the injury to the muscle or nerve is indirect or by the driving inwards of a spicule of bone, only a very accurate knowledge of the anatomical relations of the structures can enable one to frame a diagnosis of the exact site of the fracture. When, however, an injury to the head leaves no evidence of a direct injury to the orbit, or when a definite

blow is struck upon some other part, as the back of the head, a rather more difficult problem is presented for solution. A fracture may have occurred in such a case, either of one of the orbital plates, or of the base of the skull, with involvement of an ocular nerve at the base of the brain. Such a fracture will almost certainly involve the whole of a nerve, or even a group of nerves, and Panas¹ has pointed out, in regard to fractures of the base of the skull, that the sixth nerve is almost always involved on account of its anatomical relationships. Further, such fractures of the base of the skull often involve both sides, but only rarely one side. If, however, the violence be not sufficiently great to cause fracture of the skull, the brain may suffer laceration or concussion.

In cases of laceration the lesion is liable to be widespread, of course according to the extent of the cerebral injury, whilst Swanzy² thinks that concussion of the brain is quite sufficient to derange nuclear functions. Within the brain itself there are many different positions in which a lesion, be it haemorrhage or concussion, may produce a paralysis of one or more ocular muscles, and the exact localisation becomes more difficult. Fortunately, some assistance is obtained from the regularity with which symptoms are associated in cases of injury to certain parts. Thus, cortical lesions always produce associated paralysis (except in the case of the levator palpebrae),³ whilst subcortical lesions always produce a paralysis giving rise to conjugate deviations. Here again the association of symptoms may be necessary before a complete diagnosis can be made. Lower down, however, the matter becomes even more complex, for the close approximation of the nuclei for ocular muscles at the aqueduct of Sylvius and the floor of the fourth ventricle give great scope in the matter of variety of grouping. The nucleus of the sixth nerve is somewhat isolated, as is also to a less degree that of the fourth, but the various parts of the third nerve nucleus are closely packed together.

¹ Panas, *Arch. D'Ophthal.*, t. i., p. 3, 1880-81, "De la paral. du nerv. mot. ocul. ext. conseq. aux traum. du crâne." Quoted, De Wecker et Landolt, *Traité Complet d'Ophthalm.*, t. iii., p. 846, *et seq.*

² Swanzy, Norris and Oliver, *System of Diseases of the Eye*, vol. iv., p. 592.

³ Gowers, *Diseases of the Nervous System*, vol. ii., 1st edit., p. 283.

Thus, the whole of, or any part of this last nucleus, may be affected, and only incomplete knowledge of the position of the individual masses of which it is composed is as yet forthcoming to assist in solving the problem placed before us.

Briefly, so far as is known, the arrangement of the parts of the third nerve nucleus is this: That portion connected with accommodation is median in position and elongated. On each side of its anterior part lie the centres for pupillary action, as close to the middle line as they can come. Outside this, and from before backward, lie the centres for the levator palpebrae, rectus superior, lateral movements, inferior oblique, inferior rectus, and lastly, close behind this, that for the trochlearis (Fuchs).¹

From this extended line of nuclei, then, there pass through the crus groups of fibres which lie close to the pyramidal tract. These fibres emerge in bundles as the third or oculomotor nerve. Now, as to whether the lesion causing paralysis occurs in the nucleus (is nuclear) or amongst the fibres as they pass through the crus (is fascicular), it must matter comparatively little; indeed, the differentiation must be extremely difficult.

Having then reached this point, it may be well to relate the facts of the second case.

CASE II.

A young man, aged 17, was struck upon the left eye, orbital ridge, and side of the nose by the hand of a companion of his own age. The striker was walking upon the right side of the boy struck, at the time of the assault.

The patient, on being brought to the Eye Infirmary a week later, gave the following statements: He felt giddy and sick, and his nose bled freely. He staggered to a railing, but did not fall. He was never unconscious, although he was dazed. The brow and nose became very sore, and remained so during most of the night. He vomited blood clot during the night, but felt quite well in the morning and rose as usual. On going to the window he saw at once that there was something the matter, for he remarked that a woman whom he saw in the street

¹ Fuchs, *Text Book of Ophthalmology*, 2nd edit., 1900, p. 590.

also seemed to be walking on the top of a house. A doctor was called, and from him it was learned that there was no evidence of external injury, as cut or bruise to the skin, and no evidence of fracture of the orbital margin.

At the Eye Infirmary it was discovered that there was complete paralysis of the superior rectus muscle alone, and that there was a very distinct difference in horizontal level of the two eyes when the patient was directed to look straight at a distant object on the same level as the eyes. Careful examination failed to reveal any evidence of the existence of a direct injury to the orbit. There was no evidence pointing to the existence of a fracture of the base of the skull, hence it is probable that the lesion has been either nuclear or fascicular.

Subsequent visits showed that there was no tendency towards recovery after six months, but that the defect gave much less annoyance. The third case is of somewhat similar nature.

CASE III.

The patient was a healthy young man, H. M., aet. 22, who after a long and very warm day on the drill-ground at Fort Matilda became sick in the train, and whilst attempting to obtain relief fell out of the carriage window in the tunnel. He came to consciousness in Greenock Infirmary, where he found that he had had his head cut. He was unconscious for 24 hours, in bed for 72 hours, and in the infirmary only ten days. His head wound healed well, and he never had signs or symptoms of fracture of the skull. On the morning on which he came to consciousness he found that he was not able to see correctly, was seeing double indeed.

This defect was proved on investigation to be due to a complete paralysis of the left inferior rectus muscle. Careful examination failed to reveal the existence of any participating muscle. He, like the second case, became accustomed to, but did not recover from the paralysis.

Now, it may be asked, where was and what was the lesion? Had the injury in the second case been due to an orbital fracture, is it possible that the nerve twig to the superior rectus, situated, as it is, moderately deeply under the fourth

nerve, the levator palpebrae, etc., could have been injured whilst other structures escaped? Almost certainly not. Was it then at the base of the brain or at the cortex or in the subcortical region? The remarks made earlier will also answer this in the negative. Hence the lesion must have been in or near the nucleus or the fibres just coming from it. The nature of the lesion would almost surely be haemorrhagic, because, even admitting Swanzy's opinion to be correct, surely so absolute a paralysis and so localised a lesion must have been a destructive one. In the last case, also, analysis of the possible positions leads, by exclusion, to the nucleus being the most probable site of the lesion, and to haemorrhage as the cause.

Having thus devoted so much space to the consideration of the subject from a practical point of view, but little will be said regarding the literature of the subject.

The literature of this subject, then, is large, but there are very few cases recorded previously similar to those (latter) two contained here. The fact that only one muscle is affected out of a whole group controlled by one nucleus is rare. Paralysis of the iris and ciliary body as a result of trauma to the head has been recorded twice, also paralysis of the superior oblique, the abducens (most frequently), and many other muscles and groups, but in no case found has the history been more clear than in those here recorded. Simon,¹ Ahlström,² Bourgeois,³ Ginestous,⁴ and Dimmers⁵ have all made collections of cases of traumatic nuclear ocular paralysis, whilst Praun⁶ gives a very good account of this most interesting subject.

¹Simon P., "Ueber traumatische nuklear Lähmung des Augenmuskeln," Deutschmann's *Beitrag zur Augenheilk.*, B. xxiii., S. 31.

²Ahlström, Deutschmann's *Beitrag z. Augenheilk.*, B. xxiv., S. 21.

³Bourgeois, *Recueil d'Ophtal.*, 1898, p. 204.

⁴Ginestous, *Gaz. Hebdom. des Scienc. Med. de Bordeaux*, 1898.

⁵Dimmers, *Wien. Klinisch. Wochens.*, No. 10, S. 246.

⁶Praun, *Die Verletzungen des Auges.* Wiesbaden, 1899.

THE SENSORY PHENOMENA ASSOCIATED WITH HEMIPLEGIA, AS ILLUSTRATED BY TWENTY- SEVEN CASES.

By MARION J. ROSS, M.D.

THE frequency with which I have observed, during a two years' residence in a workhouse infirmary, sensory disturbance to be associated with hemiplegia has led me to pay some attention to this subject in the direction of estimating the numerical incidence of such phenomena, their detailed extent, and, if possible, something of their diagnostic importance.

So far as I can learn, little or no attention has been paid by the majority of clinicians to such sensory manifestations as may exist in a case of hemiplegia, possibly because they cannot always be demonstrated, possibly because they have not been considered of sufficient worth.

Most of the motor phenomena in connection with cerebral disease have been thoroughly investigated, and the explanation of the sequence of motor symptoms following any cerebral lesion is now more or less generally accepted. Hitzig and Fritsch in 1870 were the first to prove by experiment that definite movements of the limbs followed electrical stimulation of certain regions of the cortex. Ferrier followed their lead in 1873; and since then he, Charcot, Bastian, Dana, and numerous other observers, have attempted to localize not only the motor but the sensory centres in the cerebrum.

But while the study of motor phenomena is comparatively a simple matter the investigation of sensory lesions is beset with difficulties, and the variable, often contradictory results of the experimentalists leave much still to be done in the field of

sensory cerebral localization. Here experimental evidence is not of the same value as in motor phenomena because of the many inaccuracies which must almost of necessity arise. Gross effects resulting from equally gross lesions may be easily demonstrable to the observer, and it is possible for an animal to show quite obvious signs of local analgesia after the removal of a particular part of the cortex. But in the finer shades of sensibility where the lesion is small and the effect slight, an animal has no power wherewith to express its appreciation or otherwise of a light touch, or to describe any distinction it may draw between the various forms of sensory impressions.

In human beings also, there are many fallacies to be avoided in the testing of sensation, and many difficulties arise which it may appear at first sight almost impossible to surmount. As a *sine qua non* consciousness must be present, and a patient profoundly comatose after a cerebral apoplexy is of no value as a demonstration of anaesthesia. The hemiplegic patient must have retained or recovered at least a moderate degree of intelligence sufficient to enable him to give some verbal expression to any mental response he may make to external stimuli. And even under such favourable conditions the human mental attitude is so much a variable quantity that it entirely precludes the possibility of fixing any definite standard whereby to judge of affections of sensation. Extreme sensitiveness to external impressions, unlimited capacity for all varieties of emotion, neurotic exaggeration—these constitute a mental attitude capable of transforming the slightest tactile impression into the touch of a red-hot iron. On the other hand, dulness or apathy, cerebral exhaustion, capacity for endurance—induced either by want of perceptive power or by a more becoming stoicism—may render a patient apparently irresponsive to even a moderately severe stimulus. Again, a certain amount of power of concentration in the patient is necessary or at least acceptable to the observer, and still more so is his capability of accurate interpretation of various impressions. Then the condition of the periphery itself is of importance as regards its temperature, its hardness or softness, and its general sensitiveness.

Thus it happens that in the testing of sensation one

must necessarily depend so much on the mental acuity of the patient that the difficulty of making trustworthy observations is materially increased.

But in spite of these several drawbacks, it is, I think, possible by minutely examining the same patient under varying conditions, from day to day, to gain a fairly accurate idea of the acuity of his general sensibility, and the extent to which it has been affected by any particular lesion. This conviction led me primarily to approach the subject of sensation from a clinical point of view, and it is the results of such observations I now desire to record.

CASE I.

M. J., female, aet. 49 years.

History.—The patient had a “stroke” twenty years ago, the history of onset being that three days after her first confinement, while scrubbing a floor, she fell down unconscious. On recovering her senses she found her left arm and leg paralysed and numb, her face being drawn to the right, and her speech mumbling. Speech recovered first, and then the leg, and though eventually able to raise her arm, she could not open her hand. Personal and family history unimportant.

Examination.—Intelligence average.

Face.—Slight arcus senilis present in both eyes. No obvious facial paralysis. Speech unaffected. *Sensation.*—Over the left side of the face there is complete loss of tactile sense, and both thermal and pain sense are markedly diminished.

Special Senses.—*Sight.*—No hemianopia. Diminished visual acuity, and contracted field of vision in left eye. *Hearing.*—Watch faintly heard when laid against left ear. Heard 4 inches from right ear. *Taste.*—Totally lost on left side of tongue, the patient being tested with salt, sugar, quinine, and citric acid. *Smell.*—Totally lost in left nostril when tested with cloves, peppermint, lavender, and asafoetida.

Upper Extremity lies by the side almost quite flaccid, with the elbow flexed and the hand closed, secondary rigidity being evidenced only by resistance and pain on attempted extension of the elbow and fingers, and by exaggerated radial and deltoid reflexes. There is no power of grasp in the fingers.

Sensation.—Tactile sense is quite gone over the fingers and hand. It is present to a slight extent in the arm, but transmission is delayed and localization inaccurate. Thermal sense is absent over the hand, but the cold tube is occasionally recognized in the arm. Pain sense is also absent over the hand, and diminished in acuity in the arm. Muscular sense is gone.

Trunk.—Tactile and thermal senses are diminished over the side of the chest and abdomen. Pain sense, though present, is somewhat dulled.

Lower Extremity.—Gait is typically "paralytic," the left leg being rigid and spastic, and the toes scraping the ground with each step. Power of both flexion and extension is diminished on the left side. *Reflexes.*—Knee jerks are well marked on both sides, but especially on the left. Plantar reflex is exaggerated, and Babinski's toe phenomenon elicited on that side only.

Sensation.—Tactile, thermal, and painful senses are lost over the foot, and impaired over the leg to about the same degree as in the arm. Muscular sense is gone, the patient recognizing no position of the limb or movement of the various toes.

Diagnosis.—Left hemiplegia resulting from a lesion involving all the posterior limb of the internal capsule. The history points to embolism of the lenticulo-striate artery, but this would scarcely account for the definite affection of sensation and for the very fair motor recovery. A haemorrhage, therefore, is a most feasible supposition, though the cause for such is obscure.

CASE II.

T. S., male, aet. 60.

History.—About fifteen months ago the patient found on attempting to rise in the morning that he could not move his right leg and arm. He was conscious of a numb sensation all down the right side, but does not think his face was affected, and his speech was all right. He stayed in bed for a week, and has never been able to walk properly since, though he can move his arm fairly well. Personal and family history unimportant.

Examination.—Intelligence rather below the average.

Face.—No facial paralysis. Commencing cataracts in both eyes. *Sensation* appears perfect to all stimuli.

Special Senses.—*Sight* cannot be tested. *Hearing*.—Quite lost on right side. Watch heard when laid against left ear.

Taste.—Very markedly diminished on right side. *Smell*.—Gone on right side.

Upper Extremity.—Is not obviously affected, not being held in any fixed position, and retaining full range of movement. There is no rigidity; but the grasp is inferior to that of the left hand. *Sensation* is diminished slightly to all forms of stimuli, though localization is perfect. The patient occasionally does not know which finger is moved, and sometimes fails to appreciate a very light touch. This is most marked in the hand and forearm. Above that sensation is almost perfect.

Trunk.—Sensation somewhat dulled all over right side.

Lower Extremity.—The leg lies extended in bed, but can be drawn up and the knee flexed, though the ankle and toes cannot be voluntarily moved. The leg feels rigid, ankle clonus is easily obtained, and the knee jerks are exaggerated. *Sensation*.—Tactile sensation is much diminished, especially over the outer side of the leg and the dorsum of the foot. Thermal sense is also deficient, as regards the cold tube, though the hot is almost invariably recognized. Painful sensation is somewhat dulled, though not to the same extent as the others. Muscular sense is affected in so far as the patient does not know which toe was moved, and cannot differentiate between different weights laid on his foot and leg.

Diagnosis.—Left capsular haemorrhage, involving both motor and sensory paths in the capsule; secondary descending degeneration. General sensation is impaired, and special senses are also affected.

CASE III.

A. C., female, aet. 67.

History.—Five years ago the patient had a sudden attack of headache and vomiting, followed by paralysis of the right arm and leg. Her speech was mumbling, but there was no aphasia. She kept her bed for about a week, and then the leg recovered sufficiently to enable her to get about. The

arm gradually developed athetoid movements, which have persisted ever since.

Examination.—Intelligence good.

Face.—No paralysis. *Sensation* appears perfect.

Special Senses.—*Sight.*—No hemianopia or amblyopia, but visual acuity is defective in right eye. *Hearing.*—Diminished in right ear. *Taste.*—Gone on right side. *Smell.*—Gone on right side.

Upper Extremity hangs at the side with the third and fourth fingers extended, while the others and the thumb are flexed on the palm. Athetoid movements are marked. Voluntary movement is good. Radial reflex present. *Sensation* appears unaffected, except for a dulness in the appreciation of tactile impressions.

Trunk.—Sensation slightly improved.

Lower Extremity seems to have recovered very completely, and there is very little evidence of the paralytic gait. Knee jerk is well marked, and Babinski's toe phenomenon is present. *Sensation* appears very little affected.

Diagnosis.—Cerebral haemorrhage involving incompletely the motor and sensory part of internal capsule and contiguous optic thalamus. Here the interesting point is the involvement of the special senses to so marked a degree, while general sensation is practically unaffected.

CASE IV.

A. T., female, aet. 68.

History.—About fifteen months ago the patient became troubled with numbness of the right arm and leg, and a feeling of coldness down the right side. This lasted about six weeks, and then one evening, on trying to rise from her chair, she found she was unable to do so, and had to remain all night where she was, as she could not move her right side. She was never unconscious, but was troubled with forgetfulness of the names of things. She has never recovered the use of her limbs to any extent. *Previous History.*—About twelve years ago she had evidently an attack of acute nephritis, coming on with sickness, diminished micturition, and swelling of the face

and legs. Urine contains moderate amount of albumen. *Family History*.—Grandfather and grandmother both died of apoplexy.

Examination.—Intelligence very good.

Face.—No obvious facial paralysis. *Sensation* is defective all over the right side.

Special Senses.—*Sight*.—Amblyopia distinctly present in the right eye, evidenced by diminished visual acuity, contracted field of vision, and altered perception of colour (bright red is seen as a dirty brownish colour). *Hearing*.—Entirely gone in right ear. Watch heard at 1 inch from left ear. *Taste*.—Absent over right side of tongue. *Smell*.—Absent on right side.

Upper Extremity has no voluntary movement except a slight action of the deltoid. Rigidity is very marked and radial reflex is much exaggerated. *Sensation*.—Tactile and thermal senses are lost over hand, and defective over arm. Painful sensation is present, but impaired over arm, and especially over hand. Muscular sense is gone.

Trunk.—Sensation is affected to about the same extent as over arm.

Lower Extremity is also very rigid. Knee jerks are well marked, and a slight ankle clonus is present on both sides. *Sensation*.—Entirely gone to all stimuli over foot. Much diminished over leg and thigh.

Diagnosis.—Capsular haemorrhage involving extensively both motor and sensory paths—probably secondary to high arterial tension and arterial degeneration following nephritis. Both general and special sensations are markedly involved. The absence of aphasia is remarkable.

CASE V.

G. P., female, aet. 67.

History.—In April, 1900, the patient awoke one morning and found she could not move her right arm or leg. Her face and speech were not affected, and she did not lose consciousness. Her leg improved first, and she was able to get about, but could not use her arm much. *Personal and Family History*.—Unimportant.

Examination.—Intelligence good.

Face.—No facial paralysis. Commencing cataracts in both eyes.

Special Senses.—*Sight.*—Not tested. *Hearing.*—Watch heard only when pressed against right ear. Heard 2 inches from left. *Taste.*—Gone on right side. *Smell.*—Gone on right side.

Upper Extremity has free voluntary movement. Radial and deltoid reflexes are well marked. *Sensation* is dulled to all stimuli, especially over the hand, but muscular sense appears perfect. Cold is appreciated more than heat.

Trunk.—Sensation affected less than over arm.

Lower Extremity has also free voluntary movement. Knee jerk is well marked, but there is no ankle clonus. *Sensation* is affected to about the same extent as in arm, and muscular sense is perfect.

Diagnosis.—Left capsular haemorrhage involving sensory as well as motor path. The special senses here appear to be impaired to a greater extent than general sensation.

CASE VI.

W. P., male, aet. 39.

History.—About two years ago the patient drank very heavily for several months, and suffered from an attack of delirium tremens. On recovery he found his legs were weak, but he managed to walk with the aid of a stick. Gradually, however, he noticed a loss of power in his left arm and leg, and this was associated with a peculiar numb sensation all down the left side, and with occasional twitching of the fingers and toes, which, on these occasions, became quite blue and cold. He has also suffered occasionally from vertigo and sees objects as if they were swinging in the air. No history of syphilis.

Examination.—Patient is a very intelligent man, but is evidently nervous and has an anxious expression.

Face.—No paralysis. *Sensation* of touch, pain, and temperature are all markedly impaired, this being not quite limited by the middle line, but extending about half an inch on the right side of the forehead and chin.

Special Senses.—*Sight.*—Distinct amblyopia in left eye, consisting of contracted visual field, diminished visual acuity, and altered perception of colour. *Hearing.*—Watch heard only when pressed against left ear. Heard 2 inches from right ear. *Taste.*—Gone on left side of tongue. *Smell.*—Gone on left side.

Upper Extremity.—Movements quite free, and no obvious rigidity. Grasp is evidently diminished, and hand and arm are blue and cold. *Sensation.*—Tactile, thermal, and painful sense is diminished all over, but especially on hand. Muscular sense appears perfect.

Trunk.—Sensation diminished as on arm, the anaesthesia extending 1 inch beyond the middle line back and front.

Lower Extremity appears somewhat stiff, and there is a slight drag in walking, though voluntary movement is good. Knee jerk is well marked, but there is no ankle clonus. *Sensation* diminished to touch, pain, and temperature all over, especially in foot. Muscular sense appears perfect.

Diagnosis.—Thrombosis of the right lenticulo-optic artery, possibly specific?

CASE VII.

J. J., male, aet. 21.

History.—When aged 9 years the patient had a “stroke” while working. He suddenly lost consciousness, and on recovering his senses found his left side paralysed. He was removed to St. George’s Hospital (London), and remained there till he had regained the use of his limbs to some extent. Ever since then he has been troubled with “fits,” which came on about once a month, but occur more seldom now.

Examination.—Intelligence good. No cardiac murmur. No albuminuria.

Face.—External strabismus in right eye, with which the patient can see only very little. *Sensation* generally is diminished over the left side of the face.

Special Senses.—*Sight.*—Diminished visual acuity in left eye. *Hearing.*—Watch heard only when pressed against the left ear. Heard 3 inches from right. *Taste* lost on left side. *Smell* lost on left side.

Upper Extremity.—Movements free. No rigidity, but the arm is weaker than the right. Tactile, thermal, and painful sensations all obviously diminished. Muscular sense defective when the fingers were moved.

Trunk.—Sensation diminished all down left side.

Lower Extremity has a slight drag in walking. There is no obvious rigidity. *Sensation* affected much to the same extent as in the arm, but muscular sense appears more impaired in the lower extremity.

Diagnosis.—Embolism, with softening, involving distribution of both lenticulo-striate and lenticulo-optic artery (?). The lesion can scarcely have been cortical, on account of extensive involvement of special senses.

CASE VIII.

M. A., female, aet. 63.

History.—About three years ago the patient suddenly fell down while crossing the room, and found she could not use her right side. She gradually became unconscious, and on recovery was evidently aphasic. She has never been able to use her right arm or leg since then, but the speech has improved. *Family History.*—Grandfather, grandmother, and uncle also suffered from hemiplegia.

Examination.—Intelligence good.

Face.—Slight flattening of right side of face. Sensation is diminished to slight extent over right side.

Special Senses.—*Sight.*—Distinct hemianopia in right eye.

Hearing.—Gone on right side. Watch heard 3 inches from left

ear. *Taste.*—Gone on right side. *Smell.*—Gone on right side.

Upper Extremity.—Movements very limited. The arm feels soft, and there is a kind of athetoid movement on voluntary exertion. *Sensation* is only slightly diminished to all forms of stimuli; touch, pain, and temperature being all appreciated, but less distinctly than on the left side. Muscular sense appears perfect.

Trunk.—*Sensation* is affected here to the same extent as in arm.

Lower Extremity is very rigid, and cannot be flexed either

at the hip or knee. No knee jerk can be elicited (probably on account of extreme rigidity). *Sensation* is affected to the same extent as in arm over the thigh and leg, but is rather more defective over the foot. Muscular sense is difficult to test, but appears unaffected.

Diagnosis.—Left capsular haemorrhage, involving motor and sensory paths, the athetosis indicating involvement of contiguous optic thalamus.

CASE IX.

D. H., male, aet. 75.

History.—Four years ago the patient suddenly observed during dinner that he could not speak, and on attempting to move he found his left side was powerless and numb. He was never unconscious. He partially recovered his speech in several hours, though it was some months before he could speak distinctly. His leg improved quickly, and in a week's time he was able to walk with the help of a stick, and could move his arm to some extent.

Examination.—Intelligence fair.

Face.—No facial paralysis. Speech unaffected. *Sensation.*—Tactile, thermal, and painful sensations all diminished to about the same extent over the left side.

Special Senses.—*Sight.*—Visual acuity is diminished in the left eye. No hemianopia or contracted field of vision. *Hearing* is entirely lost on the left side. A watch placed against the right ear is heard. *Taste.*—Diminished on left side. *Smell.*—Diminished on left side. The patient could smell both lavender and asafoetida, but could not distinguish between them, except when applied to right nostril.

Upper Extremity.—The left arm is held flexed at the elbow, with the fingers closed on the palm. A fair amount of movement is present, the patient being able to extend the elbow joint to an obtuse angle. He can also open his fingers, but the movements are very slow. The grasp is good, but the fingers are with difficulty unlocked. The radial reflex is slightly exaggerated. The deltoid is not obtained. *Sensation.*—Tactile, thermal, and painful sensations are all perceived by the

patient, and his power of localization is perfect, but the sensation is much more dull than on the right side, and he cannot tell the difference between the head and the point of a pin. Muscular sense is difficult to test, as the patient can tell by pain when the arm is moved. But he cannot appreciate different weights laid on his left hand and arm.

Trunk.—Tactile, thermal, and painful sensations are affected, as in arm, over the left side.

Lower Extremity.—*Spasticity* very marked, the patient having some difficulty in moving the leg, and the movements being very slow. He cannot bend the ankle nor flex the toes. *Reflexes.*—Knee jerks very well marked on both sides, but especially on the left. On attempting to lift either leg there is a clonic spasm. Plantar reflex is exaggerated, but Babinski's toe phenomenon cannot be obtained, as the toes are already hyper-extended. *Sensation* appears affected as in arm, but the sensations are more dull, and occasionally the cold tube is not recognized when testing thermal sense. Muscular sense cannot be tested.

Diagnosis.—Haemorrhage from the lenticulo-striate branch of the right middle cerebral artery; the haemorrhage in this case has evidently extended into, and partially destroyed, the sensory fibres in the posterior third of the posterior limb of the internal capsule. The special senses are definitely involved.

CASE X.

M. H., female, aet. 33.

History.—Five years ago the patient was confined with her seventh child, and twenty-four hours after delivery she was suddenly seized with a feeling of numbness and great weight in the left leg, which gradually extended to the arm. She became unconscious, and remained so for several hours. On recovery she found she could not move her left side, and her mouth was drawn to the right. Her speech was not much affected. The leg did not recover so well as the arm, which she has been able to move freely for some time, while she can walk only with the help of a stick.

Examination.—Intelligence good.

Face.—No obvious facial paralysis. *Sensation*.—Diminished to all forms of stimuli on left side, and transmission is delayed.

Special Senses.—*Sight*.—Distinct lateral homonymous hemianopia towards right side. *Hearing* appears equal on both sides. Watch heard at a distance of 8 inches. *Taste* diminished on left side. *Smell* diminished on left side.

Upper Extremity.—Though the arm is held flexed, the patient can move it quite freely, and there does not appear to be any secondary rigidity. Radial and deltoid reflexes are not exaggerated, but the power of grasp in the fingers is less than on the right side. *Sensation*.—Tactile, thermal, and painful sensations are perceptible all over, but transmission is delayed and the sensation is dulled. Muscular sense appears perfect.

Trunk.—Sensation is perfectly perceived, though somewhat dulled.

Lower Extremity.—The left leg is rather rigid, and there is markedly diminished power of movement. The knee jerk is well marked, and plantar reflex is exaggerated. Slight ankle clonus. *Sensation* is affected more than in the arm, though the patient can still feel each variety of sensation. Localization of tactile sensibility is occasionally defective, especially over the foot; and here also heat is occasionally not recognized, though cold is always appreciated. Muscular sense appears perfect.

Diagnosis.—Haemorrhage from the right lenticulo-striate artery, involving also the posterior third of the internal capsule; and the greater part of which has been absorbed. Though this lesion was probably haemorrhagic, the cause for such haemorrhage could not be definitely ascertained. Embolism would have suggested itself as an explanation of the occurrence of hemiplegia in so young a subject, but the combination of sensory with motor symptoms renders this improbable.

CASE XI.

J. C., male, aet. 64.

History.—About a year ago, while sitting by the fire, the patient suddenly felt a peculiar sensation in his left leg, and on attempting to rise found he could scarcely move the leg.

He, however, managed, with some assistance, to walk upstairs, dragging his leg after him. His arm and his face, he says, were not affected at this time. He stayed in bed 10 days, and then managed to walk with the help of a stick. In November, 1899, he again suddenly experienced a similar numb sensation in the left leg, but this time it affected the arm also, and his whole side was powerless, though his face was not affected. He was never unconscious. *Family History*.—One brother died of “apoplexy” and one sister of rheumatism.

Examination.—Intelligence good.

Face.—Slight flattening on left side. Speech unaffected. Sensation unaffected.

Special Senses.—*Sight*.—Diminished visual acuity on left side, but no hemianopia or amblyopia. *Hearing*.—A watch is heard ticking at a distance of $2\frac{1}{2}$ inches on both sides. *Taste* diminished on left side, but present. *Smell* diminished on left side, but present.

Upper Extremity can be freely moved, but cannot be fully extended, the patient complaining of pain at the elbow when this is attempted. Radial and deltoid reflex are exaggerated. The grasp is fair, though much inferior to that on the right side. *Sensation*.—Tactile and pain sense are almost perfect over the arm and hand, though somewhat more dull than on right side. Muscular sense appears unaffected. Thermal sense is lost over almost all the arm and hand. Several times the cold tube was recognized when applied to the outer surface of the forearm, but when the hot tube was employed the patient invariably said either that it was cold or that he did not know.

Trunk.—Tactile sensation fair all over side, though occasionally not perceived on the left side of the back. Thermal sense diminished, especially with regard to the cold tube. Painful sense not much affected.

Lower Extremity can be flexed fully at hip and knee, but not at ankle. The leg appears slightly rigid. Knee jerk is well marked, and plantar reflex is exaggerated, but there is no ankle clonus. *Sensation*.—Tactile sense is present over thigh and leg, though occasionally a touch on the dorsum of the foot was referred to “the shin bone.” Thermal sense is diminished as in arm, the anaesthesia being most marked on

the dorsum of the foot. Painful sensation is only slightly affected, but more dull than on the right side. Muscular sense appears perfect.

Diagnosis.—Two separate haemorrhages into right internal capsule at an interval of a few months; the first involving the leg only—that part of motor capsule immediately contiguous to sensory path; the second, more extensive, implicating sensory as well as motor fibres.

CASE XII.

E. S., female, aet. 56.

History.—After being “out of sorts” for about a week, and complaining of sickness and dragging sensation in the head, the patient, suddenly one day three years ago, dropped her fork during dinner, and on trying to pick it up she “fainted.” On recovering consciousness she found she could not use her left arm or leg, that they felt numb, and that she could not articulate very distinctly. Her face was not affected. She never regained sufficient power to walk again, and has never since been able to use her arm. *Personal History.*—History of syphilis—in sore throat, falling out of hair, and intractable ulcer of left leg.

Examination.—Intelligence average. Patient is very emotional.

Face.—No obvious paralysis. No loss of sensation.

Special Senses.—*Sight.*—Diminished visual acuity on the left side, but no hemianopia. *Hearing.*—Equal on both sides, a watch being heard at a distance of 6 inches. *Taste.*—Diminished to some extent on the left side. *Smell.*—Diminished to some extent on the left side.

Upper Extremity lies across the chest, pronated, with the fingers bent on the palm. The deltoid action is the only voluntary movement possible. No grasp. Radial and deltoid reflexes are exaggerated. *Sensation.*—Tactile sense is present all over upper arm, though transmission is delayed. It is lost over the forearm and hand, where a light touch is not felt, except once or twice over the extensor surface of the wrist. Thermal sense is diminished all over. Painful sensation is

dulled all over. Muscular sense is also impaired, different movements not being recognized.

Trunk.—Sensation to all stimuli slightly impaired.

Lower Extremity is very rigid, and the lower third on the external surface is almost covered by an ulcer which has lasted 5 years. There are also several pale round scars on the upper part. Movements are very slow and imperfect. Knee jerk is difficult to obtain, and plantar reflex is present, but not exaggerated. *Sensation* is very difficult to test because of the scarred and ulcerated condition of the leg. But all kinds of sensation appear to be diminished to some extent.

Diagnosis.—Right capsular haemorrhage, involving sensory as well as motor path; secondary descending degeneration; cause, specific arteritis (?). Here general sensation appears impaired to a greater extent than the special senses.

CASE XIII.

J, W., male, aet. 11.

History.—About four years ago the patient began to be conscious of a weakness in his right arm. This gradually became worse, and his leg also became weaker. He occasionally suffered from headache and vomiting, and ultimately facial paralysis and nystagmus developed.

Examination.—Intelligence average. *Temperature*.—Right axilla, 100° F. Left axilla, 99·6° F.

Face.—Obvious facial paralysis on the right side. Left lateral nystagmus is marked. *Sensation* is chiefly diminished to tactile, thermal, and painful stimuli.

Special Senses.—*Sight*.—Optic neuritis. No hemianopia.

Hearing.—Diminished on the right side, where a watch is heard at a distance of 3 inches, while it is audible at 6 inches on the left. *Taste* diminished, but present on right. *Smell* diminished, but present on right.

Upper Extremity.—The hand is flexed on the forearm, while the fingers are hyper-extended. Slight athetosis is present. The movements of the arm are free, but the radial and deltoid reflexes are exaggerated, and the grasp is weak. *Sensation*.—Tactile, thermal, and painful sensations are diminished all over

the arm, and localization is defective. Muscular sense is defective.

Trunk.—Sensation is affected to rather less a degree than in the arm.

Lower Extremity.—Movements are free. Knee jerk exaggerated. Ankle clonus, knee clonus, and Babinski's toe phenomenon present on the right side. *Sensation.*—Tactile and thermal absent over foot, and painful sense is diminished. All senses are present, but impaired over leg and thigh. Muscular sense is diminished.

On March 25th the skull was trephined over the motor area, and a needle inserted in various directions. No surface tumour was detected. On examination on the 28th, sensation in the face and arm was found affected almost to the same extent as before, but was markedly defective in the leg and foot, which area in the motor cortex had been irritated by the needle.

Post-Mortem.—A large gliosarcoma was found occupying all the left internal capsule, corpus striatum, and optic thalamus. It extended to the middle line, pressing on the anterior corpora quadrigemina and veins of Galen. The ventricles were much dilated.

CASE XIV.

R. B., female, aet. 64.

History.—On waking one morning twelve years ago the patient found she was unable to use her left side, and on trying to get out of bed she lost consciousness. At first, on recovery, she could not speak, but this improved, though her articulation has since been somewhat defective. *Family History.*—Father died after a "stroke."

Examination.—Intelligence fair.

Face.—No obvious facial paralysis. Internal strabismus. *Sensation* is not affected over the face.

Special Senses.—*Sight* cannot be tested, as patient has been blind in the left eye for many years. *Hearing.*—Slightly more diminished on left than on right side. *Taste.*—Diminished on left side. *Smell.*—Diminished on left side.

Upper Extremity is held at right angles, with the fingers

flexed on the palm. The arm appears very rigid, and the radial and deltoid reflexes are exaggerated. *Sensation* is diminished over the arm and hand, in all its forms, though the cold tube is sometimes appreciated in the testing of thermal sense.

Trunk.—*Sensation* appears almost perfect.

Lower Extremity.—Rigidity is well marked. Knee jerks are well marked, and ankle clonus present on both sides, but Babinski's sign is elicited only on left side. *Sensation* is diminished to all stimuli, but only slightly over thigh and leg, and more markedly over the foot.

Diagnosis.—Right capsular haemorrhage involving both motor and sensory paths, and affecting motor speech; secondary descending degeneration.

CASE XV.

S. D., female, aet. 50.

History.—On waking one morning two years ago the patient found her left side paralysed, her mouth twisted, and her speech gone. She was not unconscious, and gradually recovered, till she was able to use her limbs fairly well.

Examination.—Intelligence fair. Heart sounds pure. No albumen in urine.

Face.—Slight flattening on left side. *Sensation* appears perfect all over.

Special Senses.—*Sight* is equal and good on both sides.

Hearing.—Entirely gone on left side, but apparently unaffected on right. *Taste* diminished on left side. *Smell* diminished on left side.

Upper Extremity.—Very good voluntary movement. Radial and deltoid reflexes exaggerated. *Sensation* appears practically unaffected.

Trunk.—*Sensation* appears practically unaffected.

Lower Extremity is somewhat rigid. Knee jerk exaggerated, and ankle clonus well marked on left side. *Sensation* appears almost perfect.

Diagnosis.—Right capsular haemorrhage involving posterior third to slight extent.

Here the special senses are more or less impaired, hearing being lost on the affected side, but normal on the sound side.

CASE XVI.

A. H., female, aet. 56.

History.—About three years ago the patient began to be aware of a numbness on her right side, and one day when walking across the floor she fell. She did not lose consciousness, but found she could not speak, and that her right side was powerless. Her speech gradually improved, and she recovered almost completely the use of her limbs.

Examination.—Intelligence good. Urine contains albumen.

Face.—No facial paralysis. *Sensation* perfect.

Special Senses.—*Sight* equal and good in both eyes. *Hearing* equal and good on both sides. *Smell* diminished on right side. *Taste* diminished on both sides.

Upper Extremity.—Voluntary movements perfect. No obvious rigidity save in exaggerated radial and deltoid reflexes. Grasp inferior to left. *Sensation* appears very little affected.

Trunk.—*Sensation* appears very little affected.

Lower Extremity has a slight drag in walking. Knee jerk is well marked. *Sensation* appears almost perfect.

Diagnosis.—Left capsular haemorrhage; involvement of sensation evidenced only by partial anosmia and diminished taste.

CASE XVII.

M. M., female, aet. 60.

History.—Four years ago, on coming home after a day's work, the patient suddenly perceived she could not use her left side, though she did not lose consciousness. She lay up for three months, and then recovered so far as to walk about, but she could never use her arm again. *Family History*.—Brother suffered from hemiplegia.

Examination.—Intelligence fair. Arteries very tortuous. No albumen in urine.

Face.—No facial paralysis. *Sensation* slightly impaired to touch, pain, and temperature.

Special Senses.—*Sight.*—Diminished visual acuity in the left eye. *Hearing.*—Watch heard only when pressed against left ear. Heard 2 inches from right. *Taste.*—Diminished on left side. *Smell.*—Diminished on left side.

Upper Extremity very rigid. No voluntary movement. Tactile, thermal, and painful sensation all slightly diminished, especially over the hand. Muscular sense is much impaired.

Trunk.—All sensations diminished slightly.

Lower Extremity is also rigid, and knee jerk is well marked. Very little voluntary movement. *Sensation* is diminished to all stimuli, and muscular sense is much impaired.

Diagnosis.—Right capsular haemorrhage involving both motor and sensory paths.

CASE XVIII.

M. B., female, aet. 41.

History.—About twelve months ago the patient fell down in the street, and, though she was not unconscious, she could not rise owing to loss of power on the right side. She was assisted home, remained in bed for ten days, and then gradually recovered. Her speech was defective at first, but evidently rather in the direction of difficulty in articulation than from aphasia.

Examination.—Intelligence fair. Speech somewhat slurred and hesitating.

Face.—No paralysis. Sensation slightly diminished on right side.

Special Senses.—*Sight* appears equal in both eyes. *Hearing.*—Diminished on right side. *Taste.*—Lost on right side of tongue. *Smell.*—Diminished, but present on right side.

Upper Extremity is slightly rigid, and radial and deltoid reflexes are well marked. Grasp is inferior to that of left hand. *Sensation.*—Tactile sensation absent over hand and diminished over arm. Painful sensation is present all over, but impaired. Thermal sense is affected in that the patient feels both hot and cold tubes as cold. Muscular sense is defective.

Trunk.—All sensations are definitely impaired.

Lower Extremity is rather rigid, and knee jerks are well marked. Distinct drag in walking. *Sensation* is affected as in arm, but to a greater extent. Tactile, thermal, and painful senses are all absent over the foot, and much diminished over the thigh and leg. Muscular sense is diminished.

Diagnosis.—Left capsular haemorrhage involving sensory as well as motor path; partial recovery—descending degeneration.

CASE XIX.

M. R., female, aet. 58.

History.—About fifteen months ago the patient had her right arm amputated for “blood poisoning,” and six weeks after the operation she suddenly lost consciousness while lying in bed. On recovery, she found she could not move her right leg, that her whole right side felt quite numb, and that she could not express herself in speech. She has never been able to use the leg since, though her speech has improved.

Examination.—Intelligence fair.

Face.—Slight right paralysis evident. *Sensation* is impaired to all stimuli, but present.

Special Senses.—*Sight*.—Diminished visual acuity in right eye. *Hearing*.—A watch is heard only when pressed against the right ear. Heard 10 inches from left. *Taste*.—Diminished on right side. *Smell*.—Diminished on right side.

Trunk.—Sensation impaired all over right side.

Lower Extremity is rather rigid. Knee jerk on the right is well marked, and ankle clonus is present. Very little voluntary movement is possible. *Sensation* is diminished to all stimuli all over, especially on the dorsum of the foot, and muscular sense is impaired.

Diagnosis.—Haemorrhage from the left lenticulo-striate artery, affecting also the sensory division of the capsule. Hearing is apparently unaffected on the sound side.

CASE XX.

M. S., female, aet. 60.

History.—About six years ago, as the patient was scrubbing a floor, she suddenly found she could not rise. After several

attempts she got up, and proceeded to bathe her left hand as it felt numb and tingling. While doing so, she fell down and remained on the floor until lifted by a neighbour. She was never unconscious, and her face does not seem to have been affected, nor was her speech, but she could not move her left arm or leg. She has been bed-ridden ever since, as her leg never recovered sufficiently for her to move about. *Family History*.—An uncle, a brother, and a sister have all suffered from “apoplexy.”

Examination.—Intelligence good.

Face.—No facial paralysis. No arcus senilis. *Sensation* appears perfect.

Special Senses.—*Sight* does not appear affected, being equal and good on both sides. *Hearing*.—Equal on both sides, but diminished in power. A watch is heard only when placed against each ear. *Taste*.—Appears perfect. *Smell*.—Appears perfect.

Upper Extremity lies at the side, supinated, with the fingers flexed on the palm. Voluntary movement is very defective, the upper arm only being movable to a slight extent. The arm can be almost fully extended on passive movement, but feels very rigid. Grasp is fair. Radial reflex exaggerated, but deltoid is not obtained. *Sensation*.—Tactile sense appears to be present to some extent all over the arm and hand, though, in the latter localization is defective, the patient invariably referring a touch on the fingers or hand to the forearm. Pain sense is also diminished, a pin prick being appreciated only when applied with some force. Thermal sense is very defective all over, the cold tube being occasionally recognized, but the warm almost never. Muscular sense also deficient, the patient recognizing that a finger is moved, but never which finger. She does not appreciate a change in position of the arm, nor are different weights perceived.

Trunk.—Sensation is rather less impaired than in the arm, but localization is defective, and occasionally a touch on the side is referred to the arm. Thermal and painful sensations are also defective.

Lower Extremity is very rigid, and there is practically no voluntary movement. Knee jerk well marked on left side,

and slight ankle clonus is obtained. Plantar reflex is exaggerated, and Babinski's toe phenomenon is elicited. *Sensation*.—Tactile present to some extent all over thigh and leg, but a touch on the dorsum of the foot is invariably referred to the lower third of the leg, and one on the sole is referred to the dorsum of the foot. Thermal sense not much affected over the thigh, but the hot tube is never recognized over the leg and foot, and the cold but seldom. Painful sense is affected as in arm. Muscular sense is defective in that patient cannot tell which toe is moved, and has no power to appreciate different weights laid on the leg or foot.

Diagnosis.—Right capsular haemorrhage involving both sensory and motor paths; secondary descending degeneration. The sensory paths being obviously involved, the very slight impairment of the special senses is remarkable.

CASE XXI.

J. C., female, aet. 55.

History.—About a year ago the patient woke one morning and found she could not move her right arm or leg, and that her whole side felt numb. She could only mumble, but could say what she wanted. Her face was not affected, and she was never unconscious. She improved in a short time, but has never quite regained the power of her right side.

Examination.—Intelligence fair.

Face.—No facial paralysis. *Sensation* appears unaffected.

Special Senses.—*Sight* equal and good in both eyes.

Hearing.—Diminished on right side: good on left. *Taste*.—Equal and good on both sides. *Smell*.—Equal and good on both sides.

Upper Extremity has free voluntary movement, though it cannot be quite fully extended. The grasp is inferior to that on the left side, and the radial and deltoid reflexes are much exaggerated. *Sensation*.—Tactile, thermal, and painful perception diminished all over hand and arm, and localization is defective. The cold tube is occasionally recognized. Muscular sense appears perfect.

Trunk.—Sensation appears almost perfect.

Lower Extremity can be freely moved. Knee jerks are exaggerated and ankle clonus obtained on both sides. Babinski's sign present in left foot. *Sensation* affected to about the same extent as in the arm, as regards perception of touch, pain, and temperature. Muscular sense appears perfect.

Diagnosis.—Left capsular haemorrhage, the lesion having involved sensory as well as motor fibres in internal capsule. Here, again, the special senses are unaffected in a lesion which has obviously implicated the power of general sensation.

CASE XXII.

W. B., male, aet. 43.

History.—Two months ago the patient suddenly fell down while walking across the room. He lost consciousness, and on recovery found he could not move his right side nor say what he wanted. The aphasia gradually improved, and he has also since regained the power of his limbs to a great extent.

Examination.—Intelligence fair.

Face.—Slight right paralysis. *Sensation* to all stimuli rather dulled on right side.

Special Senses do not appear to be affected, taste and smell being normal, while the patient can see and hear equally well on both sides.

Upper Extremity.—Fair amount of voluntary movement, no obvious rigidity, grasp less than left. *Sensation* to all stimuli is diminished over the arm, and tactile sense appears absent on the hand. Muscular sense is impaired as far as the movements of the fingers are concerned.

Trunk.—Sensation slightly impaired all over.

Lower Extremity is more rigid than arm, and knee jerks are exaggerated and ankle clonus present on both sides. *Sensation* is diminished all over as in arm, and lost to all stimuli over foot. Muscular sense appears gone.

Diagnosis.—Haemorrhage from the left lenticulo-striate artery, involving principally the leg, arm, and the sensory division of the capsule, though the nerves of special sense appear to have escaped.

CASE XXIII.

J. C., female, aet. 35.

History.—For the past five years the patient has been troubled with dyspnoea and palpitation, becoming worse from time to time, and coming on first after an attack of rheumatic fever, for which she was laid up for six weeks. A week before admission, as she was lifting a chair she suddenly fell down unconscious, and on regaining her senses found she had lost the power of her left side. The speech was not affected. She was brought to the infirmary, and since then has improved, being now able to move the leg to a certain extent.

Examination.—Intelligence good. Rough presystolic murmur leading up to a loud first sound. Reduplication of the second sound at the apex.

Face.—Slight left facial paralysis evident. *Sensation* perfect.

Special Senses.—Not affected.

Upper Extremity lies flexed across the chest, with the wrist and fingers also flexed. Secondary rigidity marked. *Sensation* perfect.

Lower Extremity lies extended and rigid. Knee jerk is not obtained, but Babinski's toe phenomenon is present.

Diagnosis.—Embolism of the lenticulo-striate branch of the middle cerebral artery affecting the anterior two-thirds of the posterior limb of the internal capsule. The posterior third is not involved.

CASE XXIV.

J. H., male, aet 45.

History.—The patient was admitted a week ago suffering from a large aneurysm of the ascending arch of the aorta, which had existed for six years, but had latterly become larger. Three days after admission he suddenly developed a right hemiplegia accompanied by motor aphasia. On examination at this time no affection of sensation was observed, and a diagnosis of embolism of the lenticulo-striate artery was made. After several days sensory symptoms began to appear, the first being sensory aphasia, which unfortunately prevented

any accurate estimation of the degree of anaesthesia. There was, however, without doubt a hemianaesthesia of the right side, and the patient gradually became comatose and died.

Post Mortem.—An embolus was indeed found in the lenticulostriate artery, and extending backwards from this to the circle of Willis; and involving the lenticulo-optic artery in passing, was a distinct thrombus, this accounting for the gradual onset of sensory symptoms after the sudden occurrence of the motor phenomena.

CASE XXV.

R. S., female, aet. 17.

History.—About two years ago the patient first noticed a tingling sensation in her left leg. This lasted about a month, and was accompanied by a feeling of weakness in the limb. The leg then began to twitch, the knee and the ankle flexing and extending, but the toes not moving. She gradually lost all power in the leg, but has since regained some degree of voluntary movement. About a year ago the left arm became affected with a similar numb sensation, and subsequent twitching and weakness. This was accompanied by a peculiar “creeping” sensation in the face, especially on the left side, and most marked near the mouth. About two months ago her tongue and mouth began to feel numb, and she could not take milk into her mouth without spilling it. At this time she could scarcely make herself understood, as she mumbled in her speech. This condition has since improved; but her eyesight has failed gradually, so that she is now totally blind. Headache and vertigo have been marked since the onset of her illness, and occasional sickness has occurred.

Examination.—Intelligence good.

Face.—Slight left facial paralysis. Sensation very little affected.

Special Senses.—Patient is totally blind from post-neuritic optic atrophy. Other senses unaffected.

Upper Extremity.—Fair amount of voluntary movement. The fingers are hyper-extended at the first phalangeal and flexed at the terminal joint. There is practically no grasp. Radial and deltoid reflexes present. *Sensation.*—Tactile sensa-

tion is appreciated all over the left arm and hand, and localization is perfect, but the feeling is more dull than on the right side. Pain and thermal sense is also diminished, but present. Muscular sense is defective. The patient occasionally knows which finger is moved, but has no idea in which position the arm is placed.

Trunk.—Sensation affected as in arm.

Lower Extremity.—Voluntary movement is good, but there is little strength in the limb. The leg gives an occasional twitch, consisting of a flexion of the knee, especially if suddenly touched on the calf. *Sensation* is affected in all forms to about the same extent as in the arm.

Operation on May 30th.—A piece of bone $2\frac{1}{2}$ inches in diameter was removed from the right parietal region, and the dura was found thickened and the brain bulged through the wound. A tumour was found covered with a thin layer of cortex. The lower angle of the wound was penetrated by the finger to find the lower edge of the tumour, which was diffuse and could not be excised. The specimen removed was found to be a gliosarcoma.

On examination of the patient on June 4th there is much more distinct facial paralysis than before; and sensation, if anything, is rather more dull.

The Upper Extremity is much more markedly paralysed as to voluntary motion, the patient being unable to raise it from the bed. *Sensation*, as a whole, is much more impaired. A light touch is not felt at all over the arm and hand, and localization is often inaccurate. Pain and thermal sense are also affected, and muscular sense is gone.

The Lower Extremity does not appear to have been so much affected by the operation, the condition being practically as at the previous examination.

CASE XXVI.

B. T., female, aet. 58, was admitted with what was found later to be a depressed fracture of the left parietal bone. A few days after admission she complained of numbness of the right leg, but on examination no defect of sensation could be

made out. She gradually lost the power of voluntary movement in the leg, and twitching began in the right arm, while her speech became indistinct, so that she called "leg" "edge." On examination of sensation on the affected side, I found it diminished in all its forms, but especially with regard to tactile sensibility, an ordinary touch being quite unappreciated and localization defective. In testing thermal sense the patient several times failed to recognize either heat or cold, but occasionally she appreciated the cold tube, though never the warm. Muscular sense in the arm was difficult to test, as the patient had also a Colles' fracture, but in the leg it was impaired, though not absent. Pain sensation was present, but dulled. These sensory defects were most marked in the leg, to a less degree on the right side of the trunk, while sensation appeared unaffected in the face. The special senses were unaffected, but optic neuritis was present. On the following day the skull was trephined, and a spicule of bone was removed, which was found pressing down into the motor area. The condition of cutaneous sensation appeared practically unaltered after the operation, and the patient died six days later. On examination of the site of the injury septic softening was found in the ascending frontal and ascending parietal convolutions, and apparently confined to this area of the cortex.

CASE XXVII.

G. C., male, aet. 50, lead worker.

History.—After working in lead works as a labourer for more than a year without any of the usual symptoms of plumbism, the patient was suddenly seized with a numb sensation all down the left side. He then became unconscious. On recovery he was evidently suffering from lateral hemianopia, as he complained of one-sided blindness. He was sent to the infirmary, when the left leg, arm, and angle of the mouth began to twitch. This condition improved after three weeks, but has come on again, and has continued more or less ever since, the period of onset being about eighteen months ago. His right side has never been affected in any way. No albuminuria and no cardiac lesion.

Examination.—The left side of the face twitches almost continually, while the left fingers are alternately flexed and extended, and the knee is also bent and straightened. The tongue is protruded markedly toward the left and is tremulous. *Sensation* slightly diminished to all stimuli.

Special Senses.—Left lateral hemianopia marked; smell, taste, and hearing not affected.

Upper Extremity.—Light touches are not felt at all, and on heavier pressure the localization is sometimes defective. Pain sensation is dulled. The patient recognizes the cold tube immediately, but constantly fails to appreciate heat; muscular sense perfect.

Trunk.—Sensation is slightly dulled all over.

Lower Extremity.—Affected to the same degree as the arm, except that there is markedly diminished pain sense.

Diagnosis.—Cortical irritation of some kind, evidenced by the twitching, by the slight affection of general sensation and the absence of involvement of the special senses, save for the hemianopia.

In twenty-four of this series of cases of hemiplegia the lesion is presumably in the internal capsule, while in three instances the motor cortex is the part affected. I now propose to consider in detail the extent of involvement of sensibility in these cases, as regards both the nerves of special sense and those concerned with the conveyance of general sensation.

Involvement of the Olfactory Nerve.—It is generally admitted that the uncinate gyrus represents the destiny of the olfactory nerves, but as regards the decussation of the fibres on their way thither, clinical observations differ somewhat from experimental and anatomical evidence. Ferrier¹ has found by experiment that in the monkey destruction of one uncinate gyrus causes anosmia *on the same side* and has stated his belief “that there is no anatomical basis of cross connection between the olfactory bulbs and their cerebral centres.” But elsewhere² he says that though the outer root of the olfactory nerve can be traced to the subicular region of the same side,

¹ Ferrier, *Functions of the Brain*, p. 185.

² Ferrier, *British Medical Journal*, 1878, vol. i., p. 558.

yet the inner may pass to the other hemisphere, though so far it has not been traced further than the corpus striatum. Testut,¹ however, describes "crossing fibres" passing from one olfactory bulb to the temporal lobe of the opposite side; and Turner² thus epitomizes what is known of the intra-cerebral course of the olfactory fibres:

1. A certain number cross, forming the pars olfactoria of the anterior commissure.

2. Many pass to the uncinate gyrus of the same side.

3. Some fibres are to be found in the neighbourhood of the internal capsule and optic thalamus.

4. A part of the anterior commissure forms a connecting strand between the hippocampal lobes of the opposite sides.

So far as personal observation goes, I find clinical evidence to be quite in favour of at least a partial decussation of the olfactory fibres in the cerebrum, as lesions affecting the sensory division of the internal capsule cause either complete or incomplete anosmia on the side opposite the lesion. In the above 24 cases eight are found to be suffering from complete anosmia, while eleven show diminished perception of smell on the same side as the hemiplegia. In no case is there any loss or obvious diminution of the sense on the same side as the cerebral lesion, and in the numerous recorded instances of similar cases this seems also to have been the condition observed.

Ferrier³ has attempted to explain the occurrence of the anosmia on the hemiplegic side by suggesting that it may be due to the loss of common sensibility of the fifth nerve, in addition to the probable crossing of a few of the olfactory fibres through the anterior commissure. Bastian,⁴ however, states that an affection of the intra-cerebral roots of the fifth nerve probably does not affect the sense of smell at all; and he believes the anterior commissure to have important functions of some kind in the conveyance of this sense.

The question thus appears to be whether the number of olfactory fibres crossing in the anterior commissure is suffi-

¹ Testut, *Anatomy of Brain and Spinal Cord*, Ed. 1900, vol. ii., p. 723.

² Turner, *System of Medicine*, by Allbutt, vol. vi., p. 753.

³ Ferrier, *British Medical Journal*, 1878, vol. i., p. 558.

⁴ Bastian, *Paralysis: Cerebral, Bulbar, and Spinal*, p. 380.

ciently great to explain the opposite anosmia in capsular lesions. Ferrier's explanation appears to me quite inadequate, inasmuch as the complete loss of smell so frequently met with clinically is often out of all proportion to the severity of the involvement of the trigeminal. In Case III. the nerves of general sensation are practically unaffected, while the olfactory sense is entirely abolished on the same side, and this without any discoverable defect in the external nasal apparatus. In Cases II., IV., V., VII., VIII., complete anosmia is associated with a general diminution, but no loss, of common sensibility; and in Case I. only is the fifth nerve affected to so great an extent as to give any degree of feasibility to such an explanation.

From a consideration of capsular lesions, therefore, it is probable that the cortical centre for smell is situated on the side opposite to the peripheral distribution of the olfactory nerves. There appears to be forthcoming little or no satisfactory and convincing proof that an isolated lesion of one uncinate gyrus, apart from any involvement of the olfactory tract or bulb, will produce an anosmia of the same side rather than a perversion of the sense of smell (Turner, *op. cit.*). Anosmia pure and simple is evidently most commonly and most completely the result of a lesion in the sensory division of the internal capsule, thus indicating the existence therein of fibres with a definitely olfactory function. This conclusion is borne out by the fact that in hysterical hemianaesthesia, which is the result of a functional rather than of an organic change in the "sensory crossway," anosmia is included in the phenomena observed.

Thus it seems fair to conclude that though there may, and probably does, exist a direct connection between the olfactory cortex and the periphery, nevertheless the crossed fibres play the more important part in connection with the conveyance of olfactory sensation.

Involvement of the Optic Nerve.—From a consideration of the relation of the visual cortical centres to the retina, the main symptom to be expected in a lesion affecting the optic radiations or pulvinar is lateral homonymous hemianopia. This phenomenon, however, I have observed in two instances only of capsular hemiplegia. In six cases visual acuity is diminished on the affected side, and in three cases there is found crossed

amblyopia, consisting of contracted visual field, diminished visual acuity, and altered perception of colour. Von Graefe formerly held the view that absolutely unilateral lesions of the cerebrum did not cause crossed amblyopia, but rather homonymous hemianopia. Charcot,¹ however, insisted that "lesions of the cerebral hemisphere producing hemianaesthesia likewise determine crossed amblyopia"; and Bastian (*op. cit.*, p. 134) remarks that unilateral amblyopia has a distinct localizing value when it is associated with hemianaesthesia, the lesion being then in the posterior third of the internal capsule. Percival,² while admitting that crossed amblyopia may occur in connection with hemianaesthesia, attempts to explain the apparent anomaly in much the same way as Ferrier explains crossed anosmia, viz., by attributing most of the phenomena to the implication of the fifth nerve. He believes it possible, as Gowers suggests, that there is a communication between the centres of the optic and trigeminal nerves, so that any excitation of the centre of the fifth may alter the sensibility of that for vision. As an example of this he cites the amblyopia arising from dental irritation of the trigeminal. But, so far as I can learn, no communication has ever been anatomically traced between the centres for the optic and trigeminal nerves. Such a theory might be sufficient to explain the diminished visual acuity in the affected eye, but the involvement of the fifth nerve in the above series of cases is so slight as to preclude any idea of a secondary influence on the optic centres sufficient to produce amblyopia. And in the example quoted—of amblyopia following dental irritation of the trigeminal—the effect on the optic nerve is presumably an irritative one, while that in hemianaesthesia is paralytic, and it is difficult to imagine two such lesions resulting in the same peripheral manifestation.

Gowers³ suggests that the occurrence of crossed amblyopia in hysterical hemianaesthesia indicates the existence of a *functional* centre, capable of being inhibited, in which is represented the whole field of one eye, and not the half fields of

¹ Charcot, *Localization of Cerebral and Spinal Diseases*, p. 108.

² Percival, Paper read to Northumberland and Durham Medical Society in 1892.

³ Gowers, *Diseases of the Brain*, p. 24.

both eyes. Granting this, it is reasonable to argue that exactly similar peripheral evidences may be produced by pathological lesions affecting the same locality.

If, as Foster¹ says, the external geniculate bodies are connected with the maculae luteae, and fibres pass from these bodies to the internal capsule, these fibres are probably connected with the maculae. Now, it is believed that each macula is projected on to the opposite cuneus; and if this be the case, it is quite sufficient to explain the occasional occurrence of crossed amblyopia rather than hemianopia in lesions of the posterior division of the internal capsule.

Involvement of the Trigeminal or Glossopharyngeal.—In considering the affections of the sense of taste in connection with capsular lesions, I have thought it wiser to do so under such a heading, opinions being still divided as to which of these nerves is responsible for the conduction of taste impressions to their cortical destination.

Owing to the difficulty of satisfactorily testing the sense of taste, especially on the posterior part of the tongue, clinical observations lose something of their value. But in eight of the above cases I was able to demonstrate total ageusia on the hemiplegic side, and in eleven a marked diminution of the sense. In no case was there a total anaesthesia of the head and face indicating an involvement of the sensory fibres of the fifth nerve to such an extent as to produce complete ageusia in addition to the other sensory manifestations. Thus, if the trigeminal be supposed to be the special nerve of taste, it is obvious that the fibres conveying impressions of taste take a separate course on their way to the cortex from those which conduct general sensation.

Involvement of the Auditory Nerve.—Though the superior temporal lobe is generally admitted to be the cortical centre for hearing, yet it has been found that destruction of this lobe on one side apparently does not cause complete crossed deafness, but rather a marked diminution in hearing on the opposite side from, and a slighter diminution on the same side as the lesion.² This statement is borne out by a study of

¹ Foster's *Physiology*, pt. iii., p. 1166.

² Collins, *Twentieth Century Practice of Medicine*, vol. x., p. 55.

capsular lesions, in which a large proportion of patients give evidence of some impairment of auditory perception on both sides. In twelve of my cases I found diminished hearing in both ears. Of these, five suffered from complete deafness on the hemiplegic side, while seven showed a much more marked impairment of the sense than was evident on the sound side. The hearing in six of the remaining cases was diminished on the hemiplegic side alone, while in four instances only was it entirely unaffected in either ear. This would seem to suggest a bilateral representation of auditory perception to some extent, the principal cortical centre being situated on the side opposite to the peripheral distribution of the auditory nerve. It is clear that the auditory fibres travel in the posterior limb of the internal capsule, and the inference therefore is either that their decussation is not complete or that some strands of fibres cross twice on their way to the cortex.

Involvement of Nerves of General Sensation.—This comprises such nerves as convey impressions of touch, pain, temperature, and muscular sense from the periphery to the cerebrum. These nerves are affected to a greater or less extent according as the site of the lesion is capsular or cortical. If the entire posterior limb of the capsule be involved a typical hemianaesthesia results, and four cases of this kind were described by Türk as early as 1859.¹ Clinically, however, it is comparatively rare to find a quite typical hemianaesthesia, and in the above series of 24 capsular lesions, general and special sensation was affected in 16 cases only. Of the remainder, three were cases in which, with practically no impairment of general sensation, the special senses were obviously diminished in acuity. The remaining 3 cases showed diminished general sensation without any impairment of the special senses.

The variable degree to which sensibility was involved is approximately as follows:

The Sense of Temperature presented some variety in its impairment, that for heat being evidently much more readily affected than that for cold, in lesions implicating the internal capsule. In nineteen patients in whom this sense was defective, ten were cases in which the cold tube was almost invari-

¹ Sturge, *British Medical Journal*, 1878, p. 783.

ably recognized, while the hot test was not appreciated at all, even when water, warm enough to be almost painful, was used. Generally speaking, it appeared that if the hot test were correctly appreciated by the patient, the cold tube presented no difficulty, and thus the sense of temperature as a whole was but little affected, though both kinds of thermal sense might be less acutely felt than on the sound side.

Tactile Sensation appeared to be impaired to the greatest extent, being more or less implicated in the entire series of cases, though sometimes to a very slight degree. In such instances only the lightest touches were unrecognized, and the sense of localization was perfect, though transmission of impression was delayed. In the more marked cases, where an ordinary touch was unappreciated, the sense of localization was not so accurate, and such patients generally gave evidence also of some impairment of their

Muscular Sense.—This appeared to be implicated in direct ratio to the severity of the tactile anaesthesia, being entirely unaffected in the milder cases, and markedly involved in the more severe types.

Pain Sensation appeared more or less impaired in all those patients who gave evidence of tactile anaesthesia. The analgesia, however, was less obvious than the affection of touch, being practically nil in the less severe cases, and but seldom complete even in definitely anaesthetic areas. In no instance did I find any impairment of the sense of pain apart from that of touch, though there are recorded various cases illustrating such a condition.¹ Paget² believes the senses of pain and temperature to be the most readily and the most permanently affected in any lesion of the brain or cord, and he suggests that the paths for these senses lie close together entirely apart from the tract conveying impressions of touch. This, however, I have not observed, but rather that tactile and muscular senses are most intimately associated, that pain sensation to a less degree is connected with touch, and that temperature apparently is distinct from either and may be affected independently of both.

¹ *British Medical Journal*, 1882, vol. i., p. 781; *ibid.*, 1888, vol. i., p. 1008.

² Paget, *British Medical Journal*, 1889, vol. i., p. 1.

It is more than probable that each form of general sensation travels by a pathway of its own in the sensory crossway; but clinically it appears that the tracts conveying impressions of touch and of pain lie near one another, and thus a capsular lesion large enough to result in analgesia almost certainly produces in addition a marked degree of tactile anaesthesia.

It seems to be a very generally accepted theory that sensation is only temporarily affected in lesions of the internal capsule; and Osler¹ says, "In hemiplegia, disturbance of the special senses is not common"; and again,² that "Hemianaesthesia is rare in hemiplegia. Slight numbness or tingling may be present, or there may be loss of sensation after a day or two, which gradually passes off." Dana³ states that, "In a good many cases there is a slight amount of hemianaesthesia during the early stage of cerebral apoplexies, but this almost invariably disappears in a few days or weeks, and it is rare that any anaesthesia of cutaneous or muscular sense is observed." Nothnagel,⁴ on the other hand, says, "From my own experience I am inclined to believe that incomplete restoration [of sensation in hemiplegia] is oftener met with than is generally believed"; and with this opinion my observations entirely coincide. It is well known that sensory fibres resist pressure better than do motor, and thus perception of sensation appears to return after a lesion more quickly than the power of voluntary movement. But in a capsular haemorrhage, where the posterior third of the posterior limb has been involved to any extent, it does not seem possible for sensation to be entirely regained by the patient. In explanation of its partial recovery it is a feasible supposition that such fibres as are stretched or compressed may recover as the compressing blood clot absorbs, while such as are lacerated give rise to permanent impairment of sensation, and it must be seldom indeed that all escape. This fact may serve as a point of differential diagnosis between haemorrhage and embolism of the lenticulo-striate artery, and Cases XXIII. and XXIV. illustrate this in different ways.

¹ Osler, *Principles and Practice of Medicine*, p. 946.

² Osler, *Theory and Practice of Medicine* (Pepper), vol. i., p. 685.

³ Dana, *Twentieth Century Practice of Medicine*, vol. x., p. 280.

⁴ Nothnagel, *Cyclopedia of Practice of Medicine*, p. 128.

In the former there was absolutely no affection of sensation, though the hemiplegia was of comparatively recent date, and the lesion was obviously an embolus in the lenticulo-striate artery, affecting only the anterior two-thirds of the posterior limb of the capsule. In Case XXIV. an embolus was also the apparent diagnosis, but was not sufficient to explain the occurrence of such marked sensory phenomena, which suggested a wider site for the lesion and increased the gravity of the prognosis.

That sensation is involved to any extent in a hemiplegia resulting from a lesion confined to the motor cortex is denied by many authorities. Ferrier¹ believes such affection of general sensibility to be due to the involvement of the hippocampal lobe secondarily by the fibres of the cingulum. But that the Rolandic area is also the chief centre for the perception of cutaneous sensation is generally believed among American and German writers (except Meynert). In fact Munk and Schiff² suggested that the so-called motor centres were not motor at all, but that in this region were stored memories of sensation associated with certain movements which are reflexly produced when the centre is stimulated; and this corresponds to some extent to the views expressed by Bastian (*op. cit.*), who considers the Rolandic area to be the kinaesthetic centre—that is, the centre for “unconscious impressions from muscles which seem to be so intimately connected with the production of movements.”

Apart, however, from an unqualified acceptance of such a theory, a study of Cases XXV., XXVI., and XXVII. indicates at least some involvement of general sensation in an essentially motor lesion. In this connection, Case XXVI. is of most interest in that a definite affection of cutaneous sensibility was associated with a lesion demonstrated *post mortem* to be confined to the Rolandic area. Here sensation was diminished to some extent in all its forms, but the impairment was most marked in the appreciation of tactile impressions. This is so

¹ Ferrier, *Brain*, 1883, vol. vi., p. 67; also *System of Medicine* by Allbutt, vol. vii.; also Croonian Lectures, *British Medical Journal*, 1890, vol. ii.; also *Localization of Cerebral Disease*.

² *Lancet*, 1883, vol. ii., p. 822.

also in lesions affecting the posterior third of the internal capsule, and suggests that the same fibres are involved in both cases. That the anaesthesia in this case was less profound than that which would result from a capsular lesion of the same recent date is explained by the fact that in the cortical lesion only the diffuse terminations of the sensory nerves are affected, while in the capsule the lesion involves the aggregation of fibres forming the sensory crossway.

In Case XXVII., again, the irritation is evidently in the Rolandic area and is accompanied by a distinct diminution of sensibility on the affected side; and this condition I have frequently observed following as well as preceding an attack of epilepsy which had begun with twitching of the thumb or great toe.

In Case XXV. the gliosarcomatous tumour probably extended beyond the motor cortex, and so this can scarcely be cited as an example of affection of cutaneous sensation following a lesion in the Rolandic area. But that the tumour did not involve the capsule is almost certain, as the special senses were not in any way affected, except for the blindness which was evidently due to the accompanying hydrocephalus. In Case XIII., on the other hand, though the diagnosis of cerebral tumour was equally obvious, the very evident diminution of smell, taste, and hearing on the affected side indicated an involvement of the capsule, and the greater affection of general sensibility suggested also this site for the lesion.

TABLE OF

Case.	Sex.	Age.	Hemi- plegia.	EXTENT OF IMPAIRMENT OF GENERAL SENSATION.			
				Face.	Arm.	Trunk.	Leg.
1.	F.	49	Left	Much dulled	Hand lost, arm impaired	Dulled	Foot lost, leg impaired
2.	M.	60	Right	Perfect	Slightly dulled	Dulled	Much im- paired
3.	F.	67	Right	Perfect	Almost per- fect	Almost per- fect	Almost per- fect
4.	F.	68	Right	Dulled	Hand lost, arm impaired	Dulled	Foot lost, leg impaired
5.	F.	67	Right	Slightly dulled	Slightly impaired	Dulled	Slightly dulled
6.	M.	39	Left	Dulled	Dulled	Dulled	Dulled
7.	M.	21	Left	Dulled	Dulled	Dulled	Dulled
8.	F.	63	Right	Dulled	Dulled	Dulled	Dulled
9.	M.	75	Left	Slightly dulled	Dulled	Slightly dull	Dulled
10.	F.	33	Left	Slightly dulled	Dulled	Dulled	Dulled
11.	M.	64	Left	Slightly dulled	Dulled	Dulled	Dulled
12.	F.	56	Left	Perfect	Hand lost, arm impaired	Dulled	Much im- paired
13.	M.	11	Right	Perfect	Impaired	Impaired	Foot lost, leg impaired
14.	F.	64	Left	Perfect	Dulled	Dulled	Dulled
15.	F.	50	Left	Perfect	Almost per- fect	Almost per- fect	Almost per- fect
16.	F.	56	Right	Perfect	Almost per- fect	Almost per- fect	Almost per- fect
17.	F.	60	Left	Dulled	Dulled	Dulled	Dulled
18.	F.	41	Right	Dulled	Much im- paired	Dulled	Much im- paired
19.	F.	58	Right	Dulled	Dulled	Dulled	Dulled
20.	F.	60	Left	Perfect	Much im- paired	Dulled	Much im- paired
21.	F.	55	Right	Perfect	Dulled	Dulled	Dulled
22.	M.	43	Right	Dulled	Dulled	Dulled	Dulled
23.	F.	35	Left	Perfect	Perfect	Perfect	Perfect
(24.)	M.	45	Right	Dulled ?	—	—	—
(25.)	F.	17	Left	Dulled	Impaired	Impaired	Impaired
(26.)	F.	58	Right	Slightly dulled	Impaired	Impaired	Impaired
(27.)	M.	50	Left	Slightly dulled	Impaired	Impaired	Impaired

CASES.

EXTENT OF INVOLVEMENT OF SPECIAL SENSES.				Lesion.
Light.	Smell.	Taste.	Hearing.	
Amblyopia	Lost	Lost	Diminished	Capsular
Cataracts	Lost	Diminished	Diminished	Capsular
Acuity diminished	Lost	Lost	Diminished	Capsular and optic thalamus
Amblyopia	Lost	Lost	Lost	Capsular
Cataracts	Lost	Lost	Much diminished	Capsular
Amblyopia	Lost	Lost	Diminished	Capsular
Blindness	Lost	Lost	Much diminished	Capsular
Hemianopia	Lost	Lost	Lost	Capsular and optic thalamus
Acuity diminished	Diminished	Diminished	Lost	Capsular
Hemianopia	Diminished	Diminished	Equal and good	Capsular
Acuity diminished	Diminished	Diminished	Diminished	Capsular
Acuity diminished	Diminished	Diminished	Equal and good	Capsular
Optic neuritis	Diminished	Diminished	Diminished	Capsular—optic thalamus corpus striatum
Blindness	Diminished	Diminished	Diminished	Capsular
Equal and good	Diminished	Diminished	Diminished	Capsular
Equal and good	Much diminished	Diminished	Equal and good	Capsular
Acuity diminished	Diminished	Diminished	Much diminished	Capsular
Equal and good	Diminished	Lost	Slightly diminished	Capsular
Acuity diminished	Diminished	Diminished	Much diminished	Capsular
Equal and good	Equal and good	Equal and good	Much diminished	Capsular
Equal and good	Equal and good	Equal and good	Diminished	Capsular
Equal and good	Equal and good	Equal and good	Equal and good	Capsular
Equal and good	Perfect	Perfect	Perfect	Capsular (Emb.)
—	—	—	—	Capsular
Blindness	Equal and good	Equal and good	Equal and good	Cortical
Optic neuritis	Equal and good	Equal and good	Equal and good	Cortical
Left lateral hemianopia	Equal and good	Equal and good	Equal and good	Cortical?

CONCLUSION.

On reviewing the subject of sensory phenomena in hemiplegia it seems justifiable to conclude that general and special sensation is more often impaired after a capsular lesion, even when the lesion is of old standing, than is usually supposed.

Many hemiplegias, whether of capsular or of cortical origin, present such definite and unmistakable signs of motor paralysis that the more subjective sensory phenomena are apt to be overlooked. Yet these sensory manifestations are of interest and importance, not only as an aid to accurate diagnosis, but also in view of the dubiety still existing as to the locality of the sensory cortex.

Occasionally, it is true, the impairment of cutaneous sensibility is so slight that the patient can feel and localize the gentlest touch when he cannot tell the difference between the head and point of a pin, and his appreciation of each form of sensation is less acute than on the sound side. Such slight peripheral evidence of a central sensory lesion may indeed seem scarcely worth recording, yet if accuracy is to be desired in the localization of the site of a lesion the smallest deviation from the normal revealed by physical examination of a patient should be of as much importance as any well-marked defect. A diagnosis founded on the examination of motor phenomena alone, without due consideration of any existing sensory impairment, is but half the truth, and is an omission of valuable aid to both the locality and extent of the lesion. On the other hand, an opinion based on a conscientious study of such motor and sensory phenomena as are presented by the patient may be confirmed by post-mortem examination, and so lead by the accumulation of evidence to the definite determination of the paths and destiny of the various sensory nerves.

Or—as occasionally happens in the experience of all—should the autopsy reveal a condition entirely at variance with the ante-mortem diagnosis, the fact that sensation has been definitely affected in such a case, and the degree and nature of the impairment correctly recorded, may be of no small value in the localization of the sensory pathways.

The testing of sensation is a task which necessitates the expenditure of much time and infinite patience, but such expenditure is surely fully justified when we consider its aim to be the elucidation of one of the most puzzling problems in the intricate subject of cerebral localization.

SOME NOTES ON EMPYEMATA IN CHILDHOOD.

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I HAVE considered as empyemata all cases where pus was found in the pleural cavity, either before or after death, so that in this series of 50 cases, 4 cases are included where loculated collections of pus were only discovered on post-mortem examination; 3 cases where the presence of pus was shewn by the exploring needle, but where no further operative interference was required; and 3 cases where the patient was admitted moribund, and died within 24 hours. Of the 14 cases which died 7 were not operated upon.

With regard to the terms used in the following pages, *Dismissed well* means that the patient left hospital with apparently a normal lung and in good general health; *Dismissed improved* implies that the general health was good, while some local condition [dulness, retraction of chest, impaired R.M., etc.] still obtained. Failure in the general health, continued pyrexia, etc., is always *Dismissed worse*.

The bulk of the cases occurred in the wards of the Royal Hospital for Sick Children, and I am indebted to the Staff for their permission to make use of them.

TABLE I.

ALL CASES—

	AGE—Under $2\frac{1}{2}$	5	$7\frac{1}{2}$	10	$12\frac{1}{2}$ years.	Total.
Dismissed well, -	3	8	9	1	1	22
Dismissed improved, -	2	2	3	3	1	11
Dismissed worse, -	1	2	-	-	-	3
Death, - - -	8	6	-	-	-	14
	<hr/> 14	<hr/> 18	<hr/> 12	<hr/> 4	<hr/> 2	<hr/> 50

OPERATION CASES ONLY—

	AGE—Under $2\frac{1}{2}$	5	$7\frac{1}{2}$	10	$12\frac{1}{2}$ years.	Total.
Dismissed well, -	3	7	9	1	1	21
Dismissed improved, -	2	2	2	2	1	9
Dismissed worse, -	1	2	—	—	—	3
Death, - - -	3	4	—	—	—	7
	<hr/> 9	<hr/> 15	<hr/> 11	<hr/> 3	<hr/> 2	<hr/> 40

It is very striking that the mortality, as shewn above, is entirely in cases under 5 years of age. The youngest child dismissed well was aged 11 months.

Sex.—28 of these cases were males, and 22 females.

Previous health.—The condition of health prior to the illness is noted in 39 cases. In 19 the children were said to have been healthy and the onset of the illness sudden. In the remaining 20 the children were either said to have been always “delicate,” or some antecedent ill health had been recognized; 3 out of the former 19 cases died, and 7 out of the latter 20.

It appears from these figures that in slightly more than half the cases there had been some antecedent ill health. Eustace Smith’s remark that, “To have pleurisy it is almost necessary that the subject should be cachectic or ill nourished,” is perhaps rather too emphatic.

Rachitis.—This was noted in only 14 cases, and signs were present in 8 of these. Of these 8 rachitic cases 5 died, but only 1 of the 6 non-rachitic ones. A large number of the other cases must have been rachitic, but, unfortunately, there are no notes on this. [Out of 176 consecutive cases treated in the medical wards of the Royal Hospital for Sick Children, 63 or 35·8 per cent. were rachitic.]

Family history.—This is noted in 41 cases. In 31 cases it was good, and in 10 tuberculosis in some form had occurred in near relatives [parents, brothers, sisters, uncles, aunts]. Of these 10 cases, 5 were dismissed well, 1 was dismissed improved, 1 dismissed worse (probably tuberculous), and 2 died. [1 was found to be tuberculous, in the other there was no post-mortem.]

Causation.—The etiology of pleurisy is still disputed. Donkin holds that it is generally secondary—usually to a pneumonia—while Eustace Smith thinks that primary pleurisy

is rare in healthy children, but may occur in cachectic or debilitated cases. Ashby and Wright consider that pleurisy may be primary, the result of cold or injury, but that empyemata are "in a vast majority of cases" the result of a pleuro-pneumonia; and Henoch says that tuberculosis and pneumonia (croupous rather than catarrhal) are important factors in causation. In the recent discussion at the British Medical Association meeting at Portsmouth, M'Guire quoted Jenner to the effect that tuberculosis is often the cause of empyema, and with this he himself agreed. Papapanagiotu, in an interesting paper on pleurisy in young infants, considered that, out of 23 cases of pleural effusion, only 2 were primarily pleural (pus, 1; serous fluid, 1).

The bacteriology of pleural effusions has recently been investigated, and it is from this direction that one must expect further light on the subject, all the more so as it seems probable that the causation in adults is different from that in children. The microbes associated with empyema in childhood are (1) the pneumococcus, either alone or mixed with streptococci; (2) a streptococcus or a staphylococcus, either alone or together; and (3) the bacillus tuberculosis.

(1) With regard to the pneumococcus cases, in many instances the pleurisy is either primary, (*i.e.* a lung lesion is entirely absent) or the lung condition, if present, is of so slight a character that it is entirely masked by the pleural. That a primary pleural infection is possible seems *a priori* probable, especially as a pneumococcic acute arthritis has been recorded with no pulmonary infection.¹ Netter² found the pneumococcus in pure culture or with a streptococcus in 15 out of 28 empyemata, and Koplik found it in 9 out of 15. The former states that in 53·6 per cent. of cases the pneumococcus is found alone, and in 3·6 per cent. associated with a streptococcus.

(2) In this group Netter found a streptococcus alone in 17·6 per cent. of his cases, and Koplik in 3 out of 15. The latter found a staphylococcus alone in 2 cases.

(3) It is with regard to the frequency of the tuberculous

¹ Widal and Lesné, *Bull. d. l. Soc. des Hop. de Paris*, 1898, p. 394.

² Netter, *Maladies de la Plèvre, Traité de Médecine*, Paris, 1901.

group that opinion differs. Examination of the pus or culture experiments are usually negative, and even inoculation is generally unsatisfactory. Netter gives 14 per cent. as the ratio in childhood. Koplik found 1 in 15.

TABLE II.

I have examined the post-mortem records of the Royal Hospital for Sick Children, and have tabulated a series of cases in which pus in appreciable quantity was found in the pleural cavity. Of these there are 33, some of which are cases whose clinical history has been made use of elsewhere, but, in the majority of cases, the pleural condition must have had little to do with the ultimate result.

No pulmonary consolidation.—In 5 cases there was no lesion in the corresponding lung, but in 1 the other lung shewed evidences of bronchitis: in 1 there was pericarditis as well, and in 1 (a case of Hodgkin's disease) pus was present in the left pleural, the pericardial, and peritoneal cavities.

Pneumonia.—5 were cases of acute pneumonia.

Broncho-pneumonia. Septic broncho-pneumonia.—8 were cases of broncho-pneumonia, and in 2 other cases small abscesses were found in the pulmonary parenchyma, associated with broncho-pneumonia.

Pyæmia.—In 4 cases the empyema was part of a general pyæmia.

Tuberculosis.—In 9 cases the corresponding lung was tuberculous, and in 3 of these there was necrosis of the pleura. In 2 an acute broncho-pneumonia was engrafted on to the tuberculous condition.

TABLE III.

I have also tabulated the condition of the pleuræ in 63 consecutive cases where, post mortem, definite lesions of the lung were found:

Tuberculous lungs—44.

Pleura, normal, - - - -	9	
Fibrinous exudate, - - - -	4	
Adhesions, - - - -	24	
Pus, - - - -	4	[pleura necrosed in 4]
Clear fluid, - - - -	3	[pleura necrosed in 2]
Tuberculous pleura, - - - -	15	

[Papapanagiotu, in 43 cases (post mortem) of pulmonary tuberculosis under the age of eighteen months, found evidences of pleurisy in 21 :

Sero-fibrinous, - - - - -	5
Mixed, - - - - -	1
Purulent, - - - - -	3
Dry, - - - - -	12]

In 16 cases the pleura was tuberculous. [In one case the pleura was tuberculous, but not the lung.]

Pleura, normal otherwise, - - - - -	1
Fibrinous exudate, - - - - -	3
Adhesions, - - - - -	8
Pus, - - - - -	2
Clear fluid, - - - - -	2

Acute pneumonia—2.

Pleura, normal, - - - - -	1
Clear fluid, - - - - -	1

Broncho-pneumonia—17.

Pleura, normal, - - - - -	4
Fibrinous exudate, - - - - -	6
Adhesions, - - - - -	3
Pus, - - - - -	3
Clear fluid (hæmorrhagic), - - - - -	1

Table II. requires some comment. From what is shewn below, the mortality in empyema varies very considerably—as would be expected—with the pulmonary condition, and in simple uncomplicated cases is small. It is thus natural that one should find few simple cases in post-mortem records. Similarly one would expect to find more tuberculous cases in post-mortem records. [Netter gives 2·3 per cent. as the mortality in pneumococcus cases, and 25 per cent. in the other varieties. This, however, refers to adults.] Out of the 9 cases where tuberculosis of the lung was present, in 2 an acute broncho-pneumonia also obtained, and it seems probable that this had some causative influence on the pleural condition.

The relative frequency of pulmonary conditions in relation to the causation of empyemata cannot thus be fairly judged from this table, though perhaps the ratio may be more accurate with regard to the mortality. It is worth while noting that of the 7 cases associated with tuberculosis, (omitting the

broncho-pneumonic cases,) 3 had necrosis of the visceral pleura, a fact agreeing with Coats' remarks on the frequency of the connection between necrosis of the pleura and acute pleurisy in pulmonary phthisis.

In Table III. empyema was present in 9.09 per cent. of the tuberculous lungs, and in 17.6 per cent. of the broncho-pneumonic cases.

It would appear from the foregoing that tuberculosis does not play a very prominent part in the causation of empyema in childhood, and that when it is an etiological factor, the primary condition is generally advanced, and this agrees with the clinical series [see below], where only 8 per cent. were tuberculous.

Papapanagiotu found only 3 cases of empyema among 54 cases of pleurisy in infants under 18 months of age; 2 were associated with pneumonia and with broncho-pneumonia. With regard to tuberculosis, pleural effusions, he says, are not met with very often, dry pleurisy being more frequent.

Age seems to have a distinct influence in the causation of empyema. The following table shews this very markedly, 32 out of 40 pleural effusions under the age of 5 years being purulent [Gee 53 out of 78 cases]. One must suppose that the resistance to the invasion of septic bacteria is slight in early life, and becomes greater later on.

TABLE IV.

	AGE—Under 2½	5	7½	10	12½ years.	Total.
Empyema, - -	14	18	12	4	2	50
Serous effusion, -	2	6	12	18	9	47
	<hr/> 16	<hr/> 24	<hr/> 24	<hr/> 22	<hr/> 11	<hr/> 97

Side of chest involved.—The empyemata were, in 31 cases, on the left side, and in 17 cases on the right. Of Scharlau's cases, 20 were left-sided and 35 right-sided; from these figures it appears that pleurisy is about equally common on the two sides [51–52]. One case was bilateral.

TABLE V.

In the following series of cases the records are mainly clinical. The cases were as a rule only seen late in the disease,

and the early history obtainable was sometimes defective ; but in 5 cases the effusion developed under observation, following an acute pneumonia in 4, and what was probably a tuberculous condition in the 5th. In the rest, the history of the case, the physical signs on admission, the course of the disease, and, in some cases, the result of post-mortem examination, formed the grounds for the classification adopted. This, however, is necessarily defective, as follows from the above remarks, and the "bronchitis" group, in particular, should perhaps have been placed elsewhere. As, however, the evidence of any pneumonic condition was absent in these cases, they have been placed by themselves, and serve in some way to emphasize the varieties of pulmonary conditions which are met with in association with empyemata.

Cases apparently primary—24.

RESULTS—Dismissed well,	-	-	-	-	-	15
Dismissed improved,	-	-	-	-	-	7
Dismissed worse,	-	-	-	-	-	2
Died,	-	-	-	-	-	—

None died in hospital ; but of the two cases dismissed worse, one was removed in a dying condition a few days after operation. The other patient had coughed up several ounces of pus on the day prior to admission ; a rib was resected, but the cavity either already was, or became septic, and the child left hospital, after some three months' residence, with the wound unhealed and symptoms of lardaceous disease.

[Mortality, 8·3 per cent.]

Cases associated with acute pneumonia—8.

RESULTS—Dismissed well,	-	-	-	-	-	3
Dismissed improved,	-	-	-	-	-	3
Died,	-	-	-	-	-	2

In one of these cases the pus discharged through the lung, and the child was dismissed improved, without any operation having been undertaken. In another a similar result was obtained after four ounces of pus had been removed by aspiration.

Of the fatal cases, one died a fortnight after operation from pericarditis and meningitis, the empyema being well ; in the

other case, loculated collections of pus were found in the pleural cavity after death.

[Mortality, 25 per cent.]

Cases associated with bronchitis—6.

RESULTS—Dismissed well,	-	-	-	-	-	3
Dismissed worse,	-	-	-	-	-	1
Died,	-	-	-	-	-	2

The case, dismissed worse, was one whose empyema had probably originated seven months prior to admission. A rib was resected, but the cavity never closed, and six months later he contracted measles and died a fortnight afterwards. The urine latterly contained a considerable quantity of albumen.

Of the fatal cases, one died suddenly in the morning before operation, and in the other severe diarrhoea set in, and acute orchitis and parotitis preceded death.

[Mortality, 50 per cent.]

Cases associated with broncho-pneumonia—7.

RESULTS—Dismissed well,	-	-	-	-	-	1
Died,	-	-	-	-	-	6

Of the fatal cases, in 4 loculated collections of pus were found in the pleural cavity after death, one had an acute nephritis, and died three days after operation, and in the other multiple abscesses were found in the lungs. In the case which recovered, only a small quantity of pus was removed by aspiration.

[Mortality, 87·5 per cent.]

Cases associated with tuberculosis—4.

RESULTS—Dismissed worse,	-	-	-	-	-	1
Died,	-	-	-	-	-	3

The case dismissed worse was a broncho-pneumonic one, which was considered to be tuberculous in character. Two of the other cases had pyopneumothorax. [In one of these (aet. 2), as no microscopic examination was made, the diagnosis of tuberculosis must be questioned. It is included under tuberculosis, however, as I did not wish to exclude any, even questionable cases, from this class.]

Case associated with pericarditis—1.

RESULT—Dismissed improved.

It was impossible in this case to say which was the primary lesion. The pleural condition on dismissal was good, but the heart had shewed signs of failure.

It is evident from the above tables that the causation of empyemata has much to do with the ultimate result, the primary empyemata and those associated with acute pneumonia, having a small mortality as compared with those complicating broncho-pneumonia or tuberculosis.

TABLE VI.

COMPLICATIONS.—In 20 of these 50 cases some complication occurred, and in several more than one.

Pericarditis was noted in 8 cases, and recovery ensued in 3, though in 1 of these, signs of cardiac failure occurred. Of these 8 cases, 4 had left-sided effusions, and 4 right-sided effusions.

Endocarditis occurred once: recovery.

Acute meningitis was noted twice, both cases ending fatally; but both were otherwise complicated, one by pericarditis, and the other by a tuberculous cavity in the lung.

Acute peritonitis occurred in one of the pericarditis cases: death.

Acute otitis media occurred in one of the nephritis cases: dismissed improved.

Acute adenitis [testis, parotid, submaxillary glands] occurred once: death.

Albuminuria was noted in 4 cases. In 3 it was transient. In the other a renal condition was known to have obtained for some time, and the child was dismissed worse.

Nephritis occurred twice. One case was dismissed improved.

Rupture into the lung occurred 4 times. In 3 of these cases a rib was resected, and in 2 recovery ensued. In the other the wound became septic, and the child was dismissed worse. The fourth case is worthy of note: Wm. E., aet. $5\frac{8}{12}$ years, took pneumonia on Nov. 15, 1893, and the crisis occurred on the 22nd. On Nov. 29 signs of pleurisy were present, and

on Dec. 16 he suddenly began to spit up purulent material in some quantity, and signs of pneumothorax were apparent. No Tubercle bacilli were found on examination of the sputum. An exploratory puncture shewed the presence of pus in the pleural cavity, but nothing in the way of operative interference was undertaken. He was dismissed on Feb. 15, 1894, in good health, but with some impairment of the extreme base of the lung.

TABLE VII.

FATAL CASES. *Results of post-mortem examination.*

There were 11 post-mortems out of the 14 fatal cases recorded above.

SIMPLE—

Sudden death before operation, - - -	1
Pneumonia—Double loculated empyema, - -	1

TUBERCULOUS—

Pyopneumothorax [see note above], - - -	2
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SEPTIC—

Pericarditis, - - - - -	5
-------------------------	---

Also in addition

Meningitis, - - - -	1
Peritonitis, - - - -	1
Multiple abscesses in lung, -	1
Multiple abscesses in lung, - - - -	1
Orchitis, parotitis, etc., - - - -	1

Excluding the tuberculous cases, in 7 out of the 9 cases multiple septic foci were found at death. The empyemata associated with broncho-pneumonia seem particularly liable to sepsis, as this occurred in 5 out of the 6 fatal cases. (In the other there was no post-mortem examination). This is probably related to the fact that, with broncho-pneumonia, various organisms are associated [the diplococcus, B. influenza, streptococcus, staphylococcus, etc.], and the infection is often mixed.

A large number of cases of empyema end fatally. Taking all the cases recorded above, and including the “dismissed worse” cases in the death list, the total mortality was 34 per cent. In the operation cases the mortality was 25 per cent.

Scharlau, in a series of 56 cases operated upon, had a mortality of 33 per cent.

It is evident from what has gone before that the immediate prognosis of an empyema in childhood is dependent on a variety of conditions other than the physical condition of the patient at the time. What perhaps affects the prognosis to the greatest extent is an early diagnosis. A simple empyema diagnosed early, and promptly operated on usually terminates favourably, and in several of the above cases the drainage tube was removed on the fourth day, and the wound healed by the tenth. Early and free drainage has two effects: it prevents the continued absorption of septic material and it removes a possible source of septic infection of other organs, a condition which is by far the most frequent immediate cause of death.

With regard to diagnosis, one point especially has appealed to me—the value of the sense of increased resistance felt on percussion over pleural effusions. This is of special value in these cases, as the vocal phenomena on which one relies so largely in adult cases are, as a rule, inapplicable in children, and the auscultatory signs are often misleading. The only other point is the necessity for exploratory puncture in all doubtful cases of pulmonary disease—a necessity which is self-evident after one has found an empyema, which had been unrecognized during life, on the post-mortem table. The dangers of puncture are practically nil, and the result of maldiagnosis is almost invariably the death of the patient.

When recognized, an empyema should be promptly dealt with. One case of sudden death before operation is recorded above (left-sided empyema). Death in such cases is probably due to syncope, and the risk is not always in proportion to the amount of the effusion or the degree of cardiac displacement. In large effusions a considerable amount of fluid should always be drawn off at once for this reason alone, though there can be little doubt that the resection of a rib is less dangerous, if the pleural cavity contains little fluid than if it be full. Syphonage without aspiration is gradual, and permits of slow reposition of displaced organs, therein differing from the sudden evacuation and rapid reposition at operation. Death on the operating table is almost always associated with

sudden evacuation of a large quantity of fluid, generally with the patient turned on to the sound side.

In all these cases which were operated upon, resection of a rib was done.¹ Osler suggests that in the pneumococcic cases this is not always necessary, aspiration or simple incision being sufficient, though in the streptococcic cases free drainage is required. One would be unwilling to resect a rib if the condition depended on a tuberculous pneumothorax.

The difficulties of diagnosis are particularly great in cases of loculated empyemata, and here the exploring needle is especially needful, as also that the surgeon should cut down at the point where pus has been found, and not at a site of election.

As an example of the evil results of pus remaining in the pleural cavity for a long time, the following case is recorded :

Richard C. (æt. 4) had an attack of "meningitis" in March, 1888, but "recovered" in a fortnight. His side was blistered at this time by the doctor's orders. He never got quite well, and a fortnight before admission caught cold and became very ill. He was admitted into hospital on Oct. 22, and a rib was resected and the pleural cavity drained. The lung, however, failed to expand, and pus continued to discharge from the wound. In April, 1889, he contracted measles and was sent to Belvidere, where he died a fortnight later. There was latterly a considerable amount of albumen in the urine. No post mortem was obtained. In this case there seems but little doubt that the empyema dated from March, 1888, and that the lung failed to expand after seven months' compression.

¹ In one case an attempt was made to avoid thoracotomy by aspiration. This case occurred under circumstances which rendered it improbable that asepsis could be obtained.

Alec G. (æt. 9) took ill on Sept. 28th, and had to take to bed next day. On Oct. 3rd there was evidence of consolidation at the right base, and on Oct. 4th he complained a good deal of pain in the chest. On the 15th the urine was scanty, bloody, and highly albuminous, and on the 18th there was evidence of effusion into the right pleural sac. On Oct. 19th his right ear commenced to discharge. On Oct. 20th 4 oz. of pus were removed by aspiration from the right pleural cavity. The fluid never reaccumulated, and on dismissal on Dec. 31st he was in excellent health; the base of the lung had, however, not completely resolved, and slight albuminuria still persisted.

Age has a distinct influence on the result. The younger the patient the greater the danger, though in one case recorded above, a child of 11 months, recovery ensued on resection of a rib. The difficulty of diagnosis in young children, their greater susceptibility to septic infection, and their small vitality are all causal to this, though, on the other hand, the sudden rebound to health, when the cause of the illness is removed, is often very striking.

The previous health of the patient, while probably causal to some extent, naturally affects the prognosis, the results in debilitated subjects being much more unfavourable than in those in good condition at the time of operation.

With regard to the pulmonary condition obtaining, it would seem that tuberculosis is only to a small extent associated with empyema in early life [8 per cent. in these cases. Netter gives the ratio as 14 per cent. In Scharlau's cases two died from tuberculosis; eight died from pneumonia, "probably on a tuberculous basis"; but he gives no post-mortem results]. When so associated the tuberculous condition is usually advanced, and the prognosis is very grave. Empyema occurring in a healthy child, and either primary or associated with pneumonia, is a disease with a small mortality; but when associated with broncho-pneumonia the prognosis is very serious—facts probably correlated to the bacterial results quoted above. [See note, p. 316.]

Still has pointed out that in empyema in children, death is very frequently the result of secondary septic infection, and in the table given above seven out of nine cases shewed other septic foci. [Still, 20 in 28 fatal cases.] Secondary infections, however, though adding greatly to the danger of the case, are by no means necessarily fatal, as three cases covered, though complicated by pericarditis, one complicated by endocarditis and one by otitis media. In the majority, however, death results. Meningitis will probably be always fatal. Nephritis is not necessarily fatal, and transient albuminuria seems to add little to the danger.

The pus may discharge through the lung, and recovery ensue without operation. It is interesting to note that in

only one of the three cases operated upon after this had occurred, did sepsis of the pleural cavity ensue.

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THE OPERATIVE TREATMENT OF MAMMARY CARCINOMA, WITH NOTES OF A CASE IN WHICH PORTIONS OF THE AXILLARY ARTERY AND VEIN WERE REMOVED WITH ADHERENT CARCINOMATOUS GLANDS.

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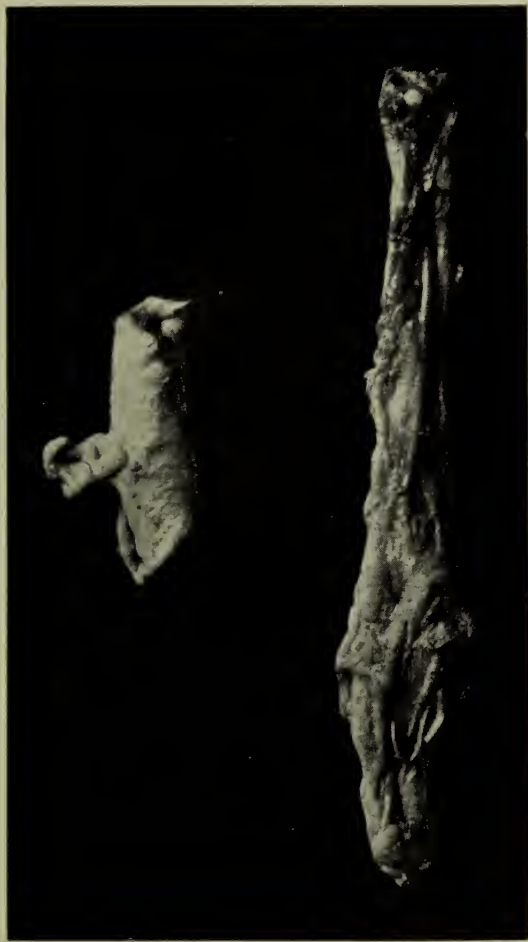
WATSON CHEYNE (Treves' *System of Surgery*, vol. ii., p. 829) in discussing the operative treatment of advanced cases of mammary carcinoma, in which the axillary vessels are involved in the glandular mass, describes the method of operation adopted by Lister. This involves amputation of the upper limb. Cheyne details a case of involvement of the vessels in which he successfully removed portions of both axillary vessels, and concludes "that, seeing it is possible to remove both axillary artery and vein without the loss of vitality of the arm, it can hardly be necessary to amputate the arm at the shoulder joint with the view of eradicating the disease."

The history of the following case lends support to Mr. Cheyne's view :

Mrs. L., aet. 60, was sent to me on June 6th, 1900, by Dr. P. Thomson. There was present in the right mamma a carcinomatous tumour of hen's egg size, adherent to the pectoralis major; and in the axilla a hard glandular mass.

At the operation I removed mamma, pectorales, and axillary contents in one mass. Dissection of the axillary vessels from the surrounding glandular mass proved difficult, and I, therefore, removed portions of $1\frac{1}{2}$ and 3 inches respectively of

FIGURE 1.



Portions of axillary artery ($1\frac{1}{4}$ inch) and vein (4 inches) excised in the course of operation for removal of mammary carcinoma. Into the lumen of the artery projects a carcinomatous bud from a carcinomatous gland, which had invaded the vessel wall.

axillary artery and vein. The accompanying illustration (Fig. I.) is a reproduction of a photograph of the portions removed.

Despite the fact that the patient was of feeble strength and constitutionally unsound, the capillary circulation in the hand was found to be fully restored ten hours after operation. The patient made a satisfactory recovery, and remains well up to the date of writing this, though it is obvious that the chances of the removal having been radical in her advanced case are less than in many.

In the matter of the treatment of earlier or favourable cases modern surgical authors agree that the statistics of recurrence after operation are becoming yearly more favourable, and that this is to be attributed largely, if not entirely, to the increasing thoroughness and magnitude of the operations performed. The investigations of Heidenhain, Stiles, and others have done much to demonstrate the need for very wide and free removal of skin, fat, and lymphatics of both mammary and axillary regions if eradication is to be attained, while gradual improvement in operative technique has rendered singularly safe an operation which involves one of the largest, if not quite the largest, wound of present-day surgery.

Whether the routine operation should include, in addition to removal of all the axillary fat and glands, removal of the pectorales is a question which, despite the able advocacy of Halsted and others, may perhaps be still regarded as *sub judice*. During the past five years I have removed the pectoral muscles in every case operated on with three exceptions. So far, experience has led me to form the following opinions:—(a) The removal of the pectorals adds little to the severity of the operation, and does not prolong the period of healing. (b) That Halsted is right when he states that the resulting disability in the arm is trivial. (c) The removal of these muscles greatly facilitates the dissection of the apex of the axilla and subclavian region. (d) By reducing the bulk of tissue to be covered removal of the pectorals permits more free removal of skin.

With reference to the comparative freedom from recurrence of the two methods, statistics are not yet sufficiently full to prove convincing. While impressed with the "thoroughness"

of the eradication possible after removal of the pectorals one has seen very complete dissections of the axilla performed without it, with gratifying results. Personally I have under observation cases in which the operation took the older, or less extensive, form in which the patients have remained free from recurrence for periods of nine and ten years respectively, and in the hands of senior surgeons are numerous cases with longer periods of freedom.

At the present time it may perhaps fairly be said that in advanced cases, such as that of Mrs. L. (*vide supra*), removal of the pectorals is essential to the completion of the operation, while in earlier cases it appears to make the assurance of complete removal doubly sure.

ADDENDUM.

While the foregoing was in the press, I operated on the following case:—Miss B., sent to me by Dr. C. Fred. Pollock. The patient is of spare build, and middle age. She suffered from a carcinoma in the inner half of the left mamma. The mass appeared to be freely movable on the chest wall, and non-adherent to the pectoralis. There were no glands tangible in the axilla.

At the operation (Feb. 3rd, 1901) the mamma was removed, and, with it, both pectoral muscles and the entire axillary contents. On raising the inner part of the pectoralis major from the ribs there were found two “beaded” lymphatic vessels running down to the intercostal spaces. The operation was, therefore, completed by the removal of portions of three ribs and costal cartilages in the region of these infected lymphatics, with as much of the periosteum and intercostal muscles as could be detached from the pericardium and pleura without opening these. (Fig. II.) The patient made a perfect recovery, the wound healing by first intention.

It may not, perhaps, be confidently hoped that the removal of these tissues has markedly brightened the prospects of this case. The removal of the pectorals did, however, reveal the involvement of the thoracic wall, which had not been suggested by the facts of the case previous to operation, and which during

FIGURE II.



Mamma, pectorales (major and minor), with portions of ribs and cartilages removed by operation for mammary carcinoma. In the inner half of the mamma is seen the carcinomatous tumour, into which an exploratory section was made during the operation. The axillary contents and the costal periosteum and intercostal muscles removed are not shown in the photograph.

the operation would almost certainly, but for the removal of these muscles, have escaped detection. The measure therefore, in this instance, assured the placing of the case in the proper statistical category.

SEQUEL TO A CASE OF ANEURYSM OF THE AORTA.

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IN the second volume of the *Glasgow Hospital Reports* (page 19) I recorded four cases illustrative of difficulties in the diagnosis of aneurysm of the aorta. The fourth of these cases was then alive, and the progress of his case had rendered the diagnosis of aneurysm more than doubtful. I am now in a position to complete the report.

The patient continued at his work in the condition described on page 37, vol. ii., until the 2nd of October, 1900, when he was suddenly seized with an extraordinarily severe pain in the left lateral region, soon followed by a brief period of unconsciousness. He was at his work at the time, and, after he regained consciousness, he was able to walk home, slowly and with assistance. The pain had to be kept in abeyance by morphia, and he could not rest in the recumbent posture. On the morning of October 5th he suddenly brought up a cupful of blood, and almost immediately expired.

Permission having been obtained to examine the thorax, Dr. Charles Workman kindly made the examination for me, and the following is an extract from his report: "On opening the thorax the left pleura is found to contain an enormous amount of blood and clot, the left lung being collapsed from the pressure, but also adherent in parts by fibrous bands from an old pleurisy. The right lung is voluminous, free from adhesions, and somewhat emphyse-

matous. The pericardium contains a small amount of slightly blood-stained fluid. The heart is considerably enlarged from hypertrophy and dilatation of the left ventricle. The aortic and pulmonary curtains are competent and healthy, and the other valvular structures also appear normal. The first part of the aortic arch is considerably dilated and very atheromatous. The innominate artery and the left carotid, though atheromatous, are normal in their arrangement. From the descending part of the arch there is a large opening leading backwards, about an inch and a half long and an inch broad, involving the left subclavian artery. This opening leads into a large saccular aneurysm, lying for the most part on the left side of the spine, and causing great erosion of the bodies of the vertebrae from the second to the sixth, and also some erosion of the posterior ends of the ribs. The aneurysm contains a large mass of very firm stratified clot, which does not nearly fill the sac. Rupture of the aneurysm has taken place into the left pleura." There was no collection of pus anywhere, and no source from which pus might have been derived, except, perhaps, the eroded vertebrae.

Here we had a clear history that carried us back to the beginning of 1894, when the aneurysm may be presumed to have originated. Throughout its course pain and dyspnoea were the most prominent symptoms, as is easily understood now that the situation of the tumour is known. But no explanation is forthcoming of the pyrexia which he presented when he left the infirmary, or of the expectoration of pus which took place thereafter. That he was able to continue at work with such a large aneurysm continuously eroding his vertebrae for such a length of time is to me a most surprising fact.

ACUTE LOBAR PNEUMONIA ; A PATHOLOGICAL AND CLINICAL STUDY OF 120 CONSECUTIVE CASES SUBJECTED TO POST-MORTEM EXAMINATION.

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IN the following paper I propose to give an analysis of the records of 120 cases of acute lobar or croupous pneumonia in which I made and recorded the post-mortem examinations. The paper will be mainly pathological in its bearings, though not entirely so, as I have examined the clinical summaries of all the cases so far as these were available to me. At present my personal observations have only to do with the facts revealed on post-mortem examination ; the clinical facts which are here analysed were supplied to me by my colleagues under whose care the cases were during life. Although acute lobar pneumonia is one of the most familiar diseases with which the practical physician is called upon to deal, the facts revealed in the post-mortem room are always of interest and frequently of great importance, as conducing to that accuracy of diagnosis and prognosis upon which all rational treatment of the disease is based. From this point of view I think the record of the personally observed facts contained in this paper may not be without interest to the readers of the present volume of the *Glasgow Hospital Reports*. I hope also that the paper may not be without value as a contribution to the already voluminous literature of acute lobar pneumonia.

For the purposes of this analysis I have, with the assistance of my house-physicians, exhaustively tabulated the information contained in all the post-mortem records of cases of acute croupous pneumonia occurring between November 1889 and November 1895, the period during which I acted as Pathologist to the Glasgow Royal Infirmary. The cases tabulated were consecutive and in no sense selected, except in so far that only cases presenting the typical morbid appearances of acute lobar or croupous pneumonia in any of its stages were included in the scrutiny of facts. Bronchopneumonia or lobular pneumonia is not included in the present enquiry. The results of this study of 120 cases of acute pneumonia I shall endeavour to formulate in a series of abbreviated tables and commentaries.

Sex.

Of the 120 cases, 94 occurred in males and 26 in females. It is unnecessary to dwell upon these figures, especially as the question of sex will also be considered in dealing with that of age. The numbers are small, but they indicate a greater prevalence of the disease in the male sex.

Age.

The age was ascertained in 112 out of the 120 cases, and the following table shows the numbers of males and females in whom the age was ascertained:

Age, known,	Males, 88	Females, 24
„ unknown, „	6	2
	<hr/> 94	<hr/> 26

The lowest age recorded in the series was 12 and the highest 80 years. As in only six cases was the age below 20, it can at once be seen that the present series of cases gives no information as to the prevalence and characters of croupous pneumonia in children. The following tables give the details of the age incidence in 88 males and 24 females, in terms of the exact age and of quinquennial periods:

TABLE SHOWING AGES OF 88 MALES WHO DIED OF
ACUTE LOBAR PNEUMONIA.

Age in Years.	Number of Cases.	Number in quinquennial periods.	Age in Years.	Number of Cases.	Number in quinquennial periods.
12	1	6	46	1	9
16	1		47	2	
17	1		48	2	
18	1		49	2	
19	2		50	2	
23	1	2	51	3	14
25	1		52	4	
26	2	9	53	2	
27	3		54	2	
29	4		55	3	
31	1	6	56	1	5
32	1		57	1	
34	1		59	1	
35	3		60	2	
36	2	14	61	2	6
37	2		62	1	
38	4		63	2	
39	3		65	1	
40	3				
41	1	15	66	1	2
42	4		68	1	
43	5				
44	3				
45	2				
			Total, 88		88

TABLE SHOWING AGES OF 24 FEMALES WHO DIED OF
ACUTE LOBAR PNEUMONIA

Age in Years.	Number of Cases.	Number in quinquennial periods.	Age in Years.	Number of Cases.	Number in quinquennial periods.
20	2	2	50	1	1
26	2	2	52	2	2
32	1	3	56	1	2
34	2		60	1	
37	1	3	65	1	1
38	2		68	1	2
41	1	5	70	1	
42	2		80	1	1
44	1				
45	1				
			Total, 24		24

These tables are interesting as showing that, in the present series of consecutive cases of acute lobar pneumonia subjected to post-mortem examination over a continuous period of six years, the greatest fatality occurred between the ages of 35 and 55. In the males 23 and in the females 7 deaths occurred up to the age of 35 years; 52 males and 11 females died between 36 and 55; in the males 13 and in the females 6 deaths occurred after the age of 55. The figures also show that at these three different age periods the mortality is much the same in males and in females. On the whole, however, the mortality in males between 36 and 55 is greater than in females; comparing the two sets of figures the number of female deaths should be 14 instead of 11 if an exact proportion had been maintained. In the earlier and later periods the death rate is slightly greater in the females than in the males; to maintain an exact proportion the number of female deaths in these periods should only have been 6.2 and 3.5 instead of 7 and 6 as seen in the tables.

This examination of sex and age shows that males are more liable to acute lobar pneumonia than females, and that in the middle period of life the mortality is greater in the male sex.

Occupation.

Of the 120 cases the occupation was recorded in 108—viz., in 86 males and 22 females. The tables on page 338 show the details of the occupations of the males and females respectively, and the classification of the occupations into indoor, outdoor, and combined indoor and outdoor occupations.

A scrutiny of these tables gives us some interesting information. While they show us that acute lobar pneumonia is in no sense an occupation disease, *i.e.* it may attack all classes of workers, they also show that the disease is very decidedly more prone to attack those whose work exposes them to the vicissitudes of the weather. Thus we find in the case of 86 males exactly 50 per cent. were engaged in work which could be classed as outdoor, and if to this we add those whose labour was partly outdoor we get nearly 75 per cent. who were exposed to the influence of the weather. If we examine the

TABLE SHOWING OCCUPATIONS OF 108 PATIENTS WHO DIED OF ACUTE LOBAR PNEUMONIA.

MALES—86 CASES.		
Baker, - - - 2	Fireman, - - - 1	Plumber, - - - 1
Blacksmith, - - - 1	French-polisher, - - 1	Potter, - - - 1
Boilermaker, - - - 1	Fruiterer, - - - 1	Quarryman, - - - 1
Brassfinisher, - - - 2	Glasscutter, - - - 1	Railway porter, - - 2
Bricklayer, - - - 1	Hawker, - - - 1	Sailmaker, - - - 1
Canvasser, - - - 1	Horseshoer, - - - 1	Sailor, - - - 1
Carrier, - - - 1	Ironworker, - - - 1	Sawyer, - - - 1
Chemical-worker, - - 2	Joiner, - - - 1	Schoolboy, - - - 1
Clerk, - - - 2	Labourer, - - - 19	Shipwright, - - - 1
Coachman, - - - 1	Lapidary, - - - 1	Shoemaker, - - - 1
Contractor, - - - 1	Mechanic, - - - 3	Slater, - - - 1
Carrier, - - - 1	Miner, - - - 5	Tailor, - - - 1
Dealer, - - - 1	Mirror-finisher, - - 1	Traveller, - - - 1
Dock labourer, - - - 1	Moulder, - - - 2	Van driver, - - - 4
Draper, - - - 2	Packer, - - - 1	Warehouseman, - - - 1
Dyer, - - - 2	Painter, - - - 1	
Engineer, - - - 2	Plasterer, - - - 1	Total, 86
Engraver, - - - 1		
FEMALES—22 CASES.		
Dealer, - - - 1	Millworker, - - - 1	Weaver, - - - 2
Dressmaker, - - - 1	Washerwoman, - - 3	—
Housewife, - - - 14		Total, 22

TABLE SHOWING OCCUPATIONS OF MALE PATIENTS, CLASSIFIED AS INDOOR, OUTDOOR, AND COMBINED INDOOR AND OUTDOOR, AND ALSO NUMBER OF PATIENTS IN EACH CLASS.

Indoor—17.	Outdoor—17.	Combined—16.
Baker, - - - 2	Bricklayer, - - - 1	Blacksmith, - - - 1
Brassfinisher, - - - 2	Canvasser, - - - 1	Boilermaker, - - - 1
Clerk, - - - 2	Carrier, - - - 1	Chemical-worker, - - 2
Draper, - - - 2	Coachman, - - - 1	Carrier, - - - 1
Dyer, - - - 2	Contractor, - - - 1	Dealer, - - - 1
Engraver, - - - 1	Dock labourer, - - - 1	Engineer, - - - 2
French-polisher, - - 1	Hawker, - - - 1	Fireman, - - - 1
Fruiterer, - - - 1	Labourer, - - - 19	Horseshoer, - - - 1
Glasscutter, - - - 1	Miner, - - - 5	Ironworker, - - - 1
Lapidary, - - - 1	Quarryman, - - - 1	Joiner, - - - 1
Mirror-finisher, - - 1	Railway porter, - - 2	Mechanic, - - - 3
Potter, - - - 1	Sailor, - - - 1	Moulder, - - - 2
Sailmaker, - - - 1	Sawyer, - - - 1	Plumber, - - - 1
Schoolboy, - - - 1	Shipwright, - - - 1	Packer, - - - 1
Shoemaker, - - - 1	Slater, - - - 1	Painter, - - - 1
Tailor, - - - 1	Traveller, - - - 1	Plasterer, - - - 1
Warehouseman, - - - 1	Van driver, - - - 4	—
22	43	21

table showing the occupation of the females attacked, we find that none of them were engaged in work which could be described as outdoor. While, therefore, it must be admitted that exposure is a frequent factor in the etiology of acute lobar pneumonia, it is at the same time clearly evident that exposure is not the only or even the most important element in the causation of the disease.

Season.

The season of the year is naturally regarded as an important etiological factor in the case of acute lobar pneumonia, and the following table gives the number of cases subjected to post-mortem examination in each month of the six years over which the present enquiry extends.

TABLE SHOWING THE NUMBER OF CASES OCCURRING IN EACH MONTH FROM NOVEMBER 1889 TO NOVEMBER 1895.

	1889	1890	1891	1892	1893	1894	1895	Total.
January, - - -		4	1	2	1	6	2	16
February, - - -			2	1	1		2	6
March, - - -		2	2	4	4	2	4	18
April, - - -			2		2	1	4	9
May, - - -		3	3	3	2		2	13
June, - - -		1	3	1	4	2	3	14
July, - - -			3	1		2		6
August, - - -		1	1		1		1	4
September, - -		2	1					3
October, - - -				1	3	2		6
November, - -	1		4	1	1			7
December, - -	4	3	3	3	4	1		18
	5	16	25	17	23	16	18	120

This table shows that over the period of six years with which this paper deals, the greatest number of cases occurred in the months of January, March, and December, and that the months of May and June come next as regards the frequency of pneumonia. Pneumonia, as judged by these post-mortem

records, is not a disease which by any means confines itself to the winter months, but occurs at all periods of the year. The months of August and September are those in which in the present series the fewest cases occurred, viz., 4 and 3 respectively for the whole period of six years. Acute lobar pneumonia may occur at any period of the year. The influence of season, like that of occupation, is an important, but evidently not the specific or determining factor in the causation of the disease.

Site of the Lesion in the Lungs and Lobes of the Lungs.

The records were carefully scrutinized with the object of ascertaining the frequency with which the local lesion of acute lobar pneumonia manifested itself in one or other lung, and in the different lobes of the lungs. The following tables show at a glance the results of this part of the enquiry:

TABLE SHOWING THE LUNG AFFECTED IN 120 CASES.

Right Lung alone involved,	-	-	-	-	62 cases.
Left Lung alone involved,	-	-	-	-	42 „
Both Lungs involved,	-	-	-	-	16 „
Total,					120

TABLE SHOWING LOBE OR LOBES AFFECTED IN 120 CASES.

Right Lung (62 cases).

Upper Lobe,	-	-	-	-	-	affected 14 times.
Middle Lobe,	-	-	-	-	-	„ 3 „
Lower Lobe,	-	-	-	-	-	„ 18 „
Upper and Middle Lobes,	-	-	-	-	-	„ 3 „
Middle and Lower Lobes,	-	-	-	-	-	„ 6 „
Upper and Lower Lobes,	-	-	-	-	-	„ 4 „
All the Lobes,	-	-	-	-	-	„ 14 „

Left Lung (42 cases).

Upper Lobe,	-	-	-	-	-	„ 11 „
Lower Lobe,	-	-	-	-	-	„ 23 „
Both Lobes,	-	-	-	-	-	„ 8 „

Both Lungs (16 cases).

Right Lung.

Upper Lobe,	-	-	-	-	-	„ 6 „
Middle Lobe,	-	-	-	-	-	„ 1 „
Lower Lobe,	-	-	-	-	-	„ 6 „
All the Lobes,	-	-	-	-	-	„ 3 „

It is thus seen that in rather more than 50 per cent. of the present series of cases grey hepatization was the condition found at the post-mortem examination, and if we add the 16 cases in which red and grey hepatization were found together the percentage is considerably increased. It is also interesting to note that death may occur during the very earliest stage of the pneumonic process.

The Day of Death.

From an examination of the clinical records which were accessible to me it was possible to fix the day of death with tolerable accuracy in 74 of the 120 cases at present under review. The details are given in the subjoined table:

TABLE SHOWING DAY OF DEATH IN 74 CASES.

3rd day of disease,	-	-	-	-	-	-	1 case.
4th	"	"	-	-	-	-	6 cases.
5th	"	"	-	-	-	-	7 "
6th	"	"	-	-	-	-	7 "
7th	"	"	-	-	-	-	14 "
8th	"	"	-	-	-	-	9 "
9th	"	"	-	-	-	-	8 "
10th	"	"	-	-	-	-	9 "
11th	"	"	-	-	-	-	4 "
12th	"	"	-	-	-	-	4 "
13th	"	"	-	-	-	-	0 "
14th	"	"	-	-	-	-	2 "
15th	"	"	-	-	-	-	0 "
16th	"	"	-	-	-	-	0 "
17th	"	"	-	-	-	-	0 "
18th	"	"	-	-	-	-	1 "
19th	"	"	-	-	-	-	2 "
Total,							74 "

The greatest number of cases p. diem died on the seventh day, which is generally accepted as the most frequent day for the crisis in pneumonia.

Day of Death and Stage of Disease.

The following table shows the stage of the pneumonic process at the time of death in the 74 cases in which it was possible from the clinical records to fix with accuracy the day of death. In one or two cases the result of this inquiry is not

such as would *prima facie* be expected, but after giving the table I think such cases may be reasonably accounted for:

TABLE SHOWING THE STAGE OF THE PNEUMONIA
AT THE TIME OF DEATH IN 74 CASES.

Stage of Disease.	Day of Death.	Number of Cases.
<i>Splenization,</i> - - -	9th day.	1 case.
<i>Red hepatization,</i> - - -	3rd "	1 "
	4th "	2 cases.
	5th "	4 "
	7th "	3 "
	8th "	1 case.
	10th "	2 cases.
	14th "	1 case.
<i>Grey hepatization,</i> - - -	4th "	4 cases.
	5th "	3 "
	6th "	6 "
	7th "	10 "
	8th "	5 "
	9th "	3 "
	10th "	5 "
	11th "	2 "
	12th "	2 "
<i>Red and grey hepatization,-</i>	6th "	1 case.
	7th "	1 "
	8th "	2 cases.
	9th "	2 "
	10th "	2 "
	11th "	1 case.
	12th "	2 cases.
<i>Purulent infiltration,</i> -	8th "	1 case.
	9th "	2 cases.
	11th "	1 case.
	19th "	1 "
<i>Abscess,</i> - - - -	14th "	1 "
	19th "	1 "
<i>Resolution,</i> - - - -	18th "	1 "
Total,	- -	74 cases.

On the whole, it may be admitted that this table shows a remarkably close agreement between the clinical history and the anatomical development of the disease. Red hepatization alone is rare after the seventh day of the disease, the greatest number of cases occurring before this day, viz., three on the seventh day and seven before it. Four cases of red hepatization are noted as having been present after the seventh day, but

this is probably to be explained by the difficulty often experienced of fixing the precise duration of the disease. On referring to the detailed records of the four cases of late red hepatization, I find that the case, in which this stage was present on the eighth day, was associated with acute pericarditis and acute pleurisy with effusion on the left side, and that the pneumonia was probably of later development than these accompanying conditions. As regards the two cases with red hepatization present on the tenth day, one had commenced as an acute bronchitis complicating chronic Bright's disease, and the other had supervened in the course of an attack of erysipelas of the leg. (In this case the opinion based on the naked-eye appearance of the lung was verified by microscopical examination.) The case of red hepatization noted as being present on the fourteenth day had occurred as a complication in the course of acute capillary bronchitis. It may, therefore, be admitted that these four cases do not seriously disturb the general belief that red hepatization is essentially an early phenomenon. Grey hepatization was not noted as having occurred before the fourth day of the disease, and by far the largest number of cases were found on and after the seventh day, viz., twenty-seven cases, as compared with thirteen before this day. Purulent infiltration is seen to be a decidedly late phenomenon, no case having occurred before the eighth day of illness. Abscess is a later development still. The one case of splenization in the series is noted as having been present on the ninth day of illness; on referring to the details of the case it is found that the pneumonia had complicated an acute bronchitis. In the case (a man aged 59), in which a resolved pneumonia was found, death occurred on the eighteenth day of illness from phlegmasia dolens with extensive thrombosis of the femoral veins as a complication of the original disease.

Other Lesions of the Lungs.

In every case in which the presence of pulmonary lesions, other than those characteristic of acute lobar pneumonia, was recorded in the account of the post-mortem examination, a note of such lesions was entered in a column of the detailed

analytical table necessary for the writing of this paper, and I propose now very shortly, in tabular form, to show how frequently such lesions were noted :

TABLE SHOWING FREQUENCY OF OTHER LESIONS IN THE LUNGS.

Acute pleurisy, on same side only, - - - -	28 times.
" on opposite side only, - - - -	3 "
" on both sides, - - - -	17 "
Pleural adhesions (fibrous), on same side, - - - -	6 "
" " on opposite side, - - - -	9 "
Empyema, - - - - -	Once.
Hypostatic congestion and oedema, on same side, -	4 "
" " on opposite side, -	15 "
Acute bronchial and tracheal catarrh, - - - -	6 "
Chronic bronchitis and emphysema, - - - -	26 "
Bronchiectasis, - - - - -	Once.
Tuberculosis (recent), - - - - -	4 "
Healed tuberculosis, on same side, - - - -	7 "
" " on opposite side, - - - -	5 "
" " on both sides, - - - -	Once.
Anthraxis, - - - - -	4 "
Opposite lung stated to be healthy, - - - -	27 "

This table is not without interest as showing that acute lobar pneumonia is very frequently associated with other morbid conditions of the lungs. As regards the presence of pleurisy, it must be remembered that there is always more or less inflammatory change in the pleura covering the affected portion of the lung. The figures demonstrate, however, that not unfrequently acute pleurisy is present on the opposite side from the pneumonia. On examining the cases of double pleurisy to find whether they were also associated with double pneumonia, I find that in no less than nine of these cases the pleurisy described in the report was double and the pneumonia single. If these nine cases be added then to the three in which the pleurisy was on the opposite side from the pneumonia, we have twelve cases in our 120 where the acute fibrinous pleurisy, frequently associated with fluid, could not be accounted for by direct extension from a pneumonic lesion in the lung of the same side. I think that this is one pathological observation which might be advanced in favour of

the view that acute lobar pneumonia is more likely to be a general or constitutional disease than a local affection of the lung itself. The frequency with which the presence of healed tuberculosis was recorded either in the same or the opposite lung is another point of great interest in this part of our inquiry: the condition was noted thirteen times in all. It is certainly of interest for the physician to bear in mind the possibility of the presence of chronic lesions in the lungs in dealing with cases of acute lobar pneumonia, as their presence cannot fail to have an important bearing both upon prognosis and treatment. The table given above shows that in fatal cases old fibrous pleural adhesions, chronic bronchitis and emphysema, and old tubercular scars are very frequent.

Condition of the Heart.

The state of the heart is a matter of the greatest importance to the physician in dealing with cases of acute lobar pneumonia, and in the following table I give the information which our 120 cases yielded on this important point :

TABLE SHOWING FREQUENCY OF ASSOCIATED CARDIAC LESIONS.

1. Normal,	-	-	-	-	-	-	-	43 times.
2. Dilatation of right side,	-	-	-	-	-	-	-	37 "
3. General hypertrophy and dilatation,	-	-	-	-	-	-	-	15 "
4. Simple hypertrophy of left ventricle,	-	-	-	-	-	-	-	3 "
5. Fatty degeneration or infiltration,	-	-	-	-	-	-	-	8 "
6. Fibrous transformation,	-	-	-	-	-	-	-	2 "
7. Aortic valve disease,	-	-	-	-	-	-	-	14 "
8. Aortic and mitral valve disease,	-	-	-	-	-	-	-	2 "
9. Mitral valve disease,	-	-	-	-	-	-	-	4 "
10. Pericarditis,	-	-	-	-	-	-	-	13 "
11. Adherent pericardium,	-	-	-	-	-	-	-	Once.

It is certainly noteworthy that in no less than 43 cases the condition of the heart at the post-mortem examination could be described as normal. The dilated and engorged condition of the chambers of the right side comes next in frequency, having been described 37 times in the reports. Aortic valve disease occurred 16 times, general hypertrophy and dilatation 15 times, and pericarditis 13 times. The influence of a

chronic lesion of the heart, either myocardial or endocardial, on the progress of a pneumonia is too well known and dreaded to require further remark in this place.

Weight of the Heart.

The weight of the organ was recorded in 62 of the 120 cases. The average weight in these 62 cases was just under 13 ounces, the maximum weight being 25 ounces, occurring once, and the minimum 7, occurring once. Weights of 20 ounces or over were recorded five times in all, and weights of 9 ounces and under six times in all. It would thus seem that acute pneumonia is not infrequently associated with enlargement of the heart, and anything like confirmed general hypertrophy of the organ must add gravely to the prognosis.

Acute Pericarditis.

Pericarditis was observed in 13 cases, and must always be regarded as a grave complication of the original disease. In some cases, no doubt, acute pericarditis occurring in the course of acute lobar pneumonia must be attributed to direct extension of the inflammatory process to the pericardial membrane, and that being so one would naturally expect that this complication would be most frequently associated with a left-sided pneumonia. With regard to the present series of cases this proves not to be the case, as the following table shows :

TABLE SHOWING RELATION OF ACUTE PERICARDITIS TO THE LUNG AND LOBE AFFECTED, AND TO THE STAGE OF THE DISEASE.

<i>Right Lung (8 cases).</i>						
<i>Lobe affected,</i>	{	Upper lobe,	-	-	-	3 cases.
		Middle,	-	-	-	1 case.
		Lower,	-	-	-	1 „
		Middle and lower,	-	-	-	2 cases.
		All lobes,	-	-	-	1 case.
					<hr/>	8 cases.
<i>Stage,</i>	{	Red hepatization,	-	-	-	4 cases.
		Grey hepatization,	-	-	-	4 „
					<hr/>	8 „

Left Lung (3 cases).

<i>Lobe affected,</i>	{	Upper lobe,	-	-	-	-	1 case.
		Lower,	-	-	-	-	1 „
		Both lobes,	-	-	-	-	1 „
							<hr/>
							3 cases.
<i>Stage,</i>	{	Grey hepatization,	-	-	-	-	2 cases.
		Purulent infiltration,	-	-	-	-	1 case.
							<hr/>
							3 cases.

Both Lungs (2 cases).

<i>Stage,</i>	{	Grey and red hepatization,	-	-	-	1 case.
		Purulent infiltration,	-	-	-	1 „
						<hr/> 2 cases.

Condition of the other Organs.

It is impossible within the limits of a paper like the present to enumerate the various conditions which were met with in all the other organs in our investigation of cases of acute lobar pneumonia. The state of the spleen, the liver, the kidneys, and the brain may be shortly adverted to. As regards the brain, however, it must be stated that this organ was not very frequently examined.

The Spleen.—Cloudy swelling of the spleen, with enlargement and softness of the organ was described 58 times, or in nearly 50 per cent. of the cases. The weight of the organ was ascertained and recorded 30 times, the average weight being found to be $7\frac{3}{4}$ ounces, indicating a considerable enlargement of the organ. The maximum weight was 15 ounces, the minimum 4 ounces, each occurring once. This very constant enlargement of the spleen is not without significance, and may be regarded as another anatomical evidence of the constitutional nature of the disease. The spleen was described as healthy in 15 cases.

The Liver.—The liver was the seat of cloudy swelling in 27, and of fatty infiltration in 20 cases; the parenchyma of the organ being thus affected in 47 cases. In 15 cases the organ was described as healthy.

The Kidneys.—The condition of the kidneys is certainly a most important factor in the natural history of acute lobar

pneumonia. The following table gives at a glance the condition in 62 cases in which a record was kept :

Kidneys, healthy,	-	-	-	-	-	-	16 cases.
Cloudy swelling,	-	-	-	-	-	-	24 „
Tubular nephritis,	-	-	-	-	-	-	9 „
Interstitial nephritis,	-	-	-	-	-	-	12 „
Calculus,	-	-	-	-	-	-	1 case.
							<hr/>
							62 cases.

The occurrence of acute lobar pneumonia in the course of chronic Bright's disease is well known, and in our present series of cases we find that this association was noted to have been present at least 21 times.

The Brain.—Unfortunately the head was only examined 12 times, with the following results :

Brain, healthy,	-	-	-	-	-	-	5 cases.
Cerebral softening,	-	-	-	-	-	-	2 „
Fracture of skull,	-	-	-	-	-	-	1 case.
Acute meningitis,	-	-	-	-	-	-	4 cases.
							<hr/>
							12 „

It is interesting to note that in this relatively small number of head examinations acute meningitis was discovered four times, as it is well known, both clinically and pathologically, to be a common complication of acute lobar pneumonia.

It is unnecessary further to discuss the condition of the organs in general in the present series of cases, except just to add that acute peritonitis was described three times, marked atheroma of the aorta seven times, and aneurism of the aorta twice.

The Nature of Acute Lobar Pneumonia.

Acute lobar pneumonia, both from the clinical and the pathological point of view, may be classified as primary and secondary. By primary pneumonia we mean that variety of the disease which begins acutely in the midst of ordinary health and runs the characteristic clinical course. As secondary pneumonia we class those cases in which the pulmonary lesion may be looked upon as the direct result either of some old-standing primary lesion elsewhere or of septic

absorption or insufflation. As regards the actual structural change in the pulmonary tissue, the condition, both macroscopically and microscopically, may be practically the same in each form, and I do not believe that a study of the morbid anatomy of the lung alone would enable us to make the distinction. Neither do I think it possible at present by the ordinary bacteriological methods to differentiate the primary and secondary forms of the disease, for the same germs, as Weichselbaum has, I think, conclusively shown, may be found in each. As regards the 120 cases which have now been analysed, I have no bacteriological observations to submit which would be of service in the elucidation of this point, although in several of the cases bacteriological investigations were carried out, chiefly for class and demonstration purposes. Any views I may therefore have to express on this aspect of the nature of pneumonia must be based on purely clinical and anatomical grounds.

On examining the records I find that there are only 18 cases in all, of which it could be affirmed that they were truly examples of acute secondary lobar pneumonia: 9 were septic cases, secondary to wounds or to erysipelas; 8 were insufflation pneumonias, secondary to cancerous disease of the gullet or trachea; and 1 was secondary to opium poisoning. Of course, as may be seen in the tables, there were 21 cases which were associated with Bright's disease, and these might be regarded by some as raising the total number of secondary cases to 39, but I think the lobar pneumonia intercurrent in the course of Bright's disease may fairly enough be classed as primary pneumonia. If this be admitted, then of our 120 cases 102 may legitimately be regarded as examples of acute primary lobar pneumonia.

As regards the nature of secondary pneumonia little need be said. It may be looked upon as a purely local inflammation of the lung dependent upon the action of definite morbid agents, and in no sense differing from similar inflammations similarly produced in other regions of the body. Although I have said that morbid anatomy is not capable of absolutely differentiating secondary from primary pneumonia by the naked-eye appearances of the inflamed lung alone, yet I must

modify this statement in so far as to admit that to the eye of a trained pathologist there is something about the appearance of an insufflation or a septic pneumonia which might in itself raise a suspicion as to its true nature. The inflammatory lesion has on the whole a somewhat coarse character, with here and there areas suggestive of localized pus-formation, which is different from the homogeneous and uniform appearance of the red or grey hepatization of a primary lobar pneumonia. But even the skilled pathologist might not care to base his diagnosis on the naked-eye appearances alone without taking into consideration the information to be obtained from a careful investigation of the other features of the case.

It is not so easy, however, to be so sure of the true nature of primary acute lobar pneumonia. Whether the disease is to be regarded as a local affection of the lung or as a general constitutional disorder of the nature of a specific fever is a problem not quite easy of solution. It is not my intention to enter upon an academic discussion of all the aspects of this difficult problem, which embraces the consideration of many points not included in the present investigation. My object is simply to inquire what light the 102 cases of primary acute lobar pneumonia dealt with in this paper throw upon this question. On the whole, I think that the facts demonstrated in this analysis support the view, now very generally accepted by physicians and pathologists, that primary acute lobar pneumonia is a general febrile disease, with a local lesion usually, but perhaps not quite invariably, in the lung. It may be urged that the results of the present inquiry do not carry us very far towards this solution of the problem, particularly with regard to that part of it which suggests that the local pulmonary lesion may, though I admit very rarely, be absent altogether, yet I think they do carry us a little way. The analysis shows us that no age or occupation is exempt from the disease, although no doubt, as regards occupation, a greater prevalence may be admitted in occupations which are to be classed as outdoor occupations. Such facts may perhaps be regarded as pointing to a general rather than a local disease, although it must at the same time be admitted that too great weight must not be attributed to them if we also take into

account the greater influence of outdoor work and the distinctly greater prevalence of the disease in the male sex.

The facts brought out with regard to the prevalence of acute lobar pneumonia at different seasons of the year are distinctly in favour of the view that the disease is general and not local. The figures in the table on page 339 suggest that the incidence of the disease is subject to epidemic influences, and the circumstance that, in a series of observations extending over 6 years, nearly as many cases occurred in May and June as in December and January is certainly indicative of a general rather than a local disease. Were the disease simply a local inflammation of the lung, we would expect a very much greater prevalence in the cold months of winter and early spring; in round numbers, however, we find that about 60 per cent. of the cases occurred in the winter months from October to March, and about 41 per cent. in the summer months from April to September inclusive. Such a difference, however, indicates something more than the mere effect of cold as producing a local pulmonary inflammation: the fact that 41 per cent. of the cases occurred during the summer months suggests not only epidemic influences, but also a general rather than a local morbid process.

The facts as to the morbid anatomy of acute lobar pneumonia, in so far as they have been elicited in the foregoing analysis, may also, I think, be taken as pointing in the same direction. The somewhat remarkable variation of the site of the lesion in the lung in different cases, the fact that all the lobes of a lung may be affected in some cases and both lungs in others are circumstances not without significance. Were the disease in its origin a purely local affection it might be expected to begin with more or less constancy in a particular area of the lung, for example in the apex or in the base. This, however, as a reference to the table on page 340 shows, is by no means the case, although no doubt the lesion is more frequent in the right lung and in the lower lobe. Further, the frequent association of lobar pneumonia with pleurisy of the opposite side and with pericarditis may be taken as indicative of a general rather than a local disease. The facts brought out with reference to the associated pericarditis are interesting in

this regard. We have seen that in our cases pericarditis was most frequent in right-sided pneumonias, in three of them the lesion being situated in the upper lobe, a circumstance which points to general infection rather than to mere local extension. The association of lobar pneumonia with acute peritonitis points to the same thing, particularly if, as sometimes happens, all the great serous cavities are more or less involved.

The condition of the other organs, as demonstrated in the course of this analysis, is such as is usually met with in the specific fevers. We have seen that cloudy swelling of the liver, kidneys, and spleen are very frequent associated conditions. The constancy with which enlargement of the spleen is met with is perhaps worthy of special mention in this regard. My friend, Dr. David M'Crorie, assistant physician and bacteriologist to the Glasgow Royal Infirmary, informs me that in a very large number of examinations he has never failed to obtain the pneumococcus in cultures from the spleen in cases of acute lobar pneumonia. On the whole, then, I think, it may be admitted that the facts as regards morbid anatomy brought together in this paper support the view that acute lobar pneumonia is a general and not a local disease.

One of the strongest clinical arguments in favour of this view as to the nature of acute lobar pneumonia is the constancy with which the crisis of the disease occurs on the 7th or 8th day. A reference to the table showing the day of death in 74 of our cases makes it clear that by far the greatest number of cases die on and after the seventh day of the disease. The actual figures are these: On the 6th day and before it 21 cases died; from the 7th to the 11th day inclusive 48 cases died. These figures confirm the clinical argument based on the constancy of the critical day in pneumonia, and form a fitting conclusion to the foregoing remarks, in which I have endeavoured to give expression to my opinion, based on six years' observation and study in the post-mortem room (supplemented, as regards my personal experience, by six additional years of clinical study in the wards), that acute lobar pneumonia is a general febrile disease.

SEPTIC DISEASES OF THE KIDNEY.

By DAVID NEWMAN, M.D.

SEPTIC lesions of the kidney assume many forms according to the channel through which the infective material is conveyed to the organ. The diseases may be classified either according to their cause or in relation to the part primarily involved. From a pathological point of view the former basis of classification is to be preferred, but from a clinical standpoint the latter is more useful.

Various terms are employed to designate the precise situation of the lesion. When the mucous membrane of the pelvis is attacked, without any distension of that cavity, the disease is named pyelitis; while, if pus accumulates as the result of mechanical obstruction, the condition is referred to as pyonephrosis. Again, should the inflammation extend beyond the pelvis, and attack the renal substance, forming in it independent accumulations of pus, the condition is designated pyelonephritis. Occasionally an acute suppurative inflammation of the renal substance may occur without any affection of the pelvis or the ureter; to this condition we limit the term suppurative nephritis. This very simple arrangement is doubtless convenient for every-day use, but like most clinical groupings it is liable to the imputation of being superficial and insufficient; it takes no cognizance of the various etiological factors, nor does it regard the natural history of the various maladies.

The following classification, which is based upon the mode of infection as well as the tissues involved in the septic processes, may be adopted with advantage in studying the *Pathology*.

1. *Purulent embolic nephritis*.—A descending septic and suppurative lesion of the kidney, without pre-existing disease of the conducting and collecting portions of the urinary tract, the septic virus being conveyed to the kidney by the blood.

2. *Purulent interstitial nephritis*.—An ascending interstitial nephritis, infection being by the lymphatics, from a primary septic focus in the lower urinary passages.

3. *Acute septic nephritis without suppuration*.—An ascending septic lesion of the kidney without suppuration, the virus being carried to the kidney (most commonly to the cortex) by the lymphatics.

4. *Pyelonephritis*.—Suppurative nephritis with antecedent septic disease of the pelvis, the secondary foci in the parenchyma of the kidney being mostly due to direct contamination through the uriniferous tubules and lymphatics.

5. *Pyelitis*.—Suppurative disease of the mucous membrane of the pelvis, without distension of that cavity.

6. *Pyonephrosis*.—Accumulation of pus or of purulent urine in the pelvis of the kidney, the accumulation being a result of mechanical obstruction, with atrophy of the renal tissue; but without secondary infective foci or independent accumulations of pus in the parenchyma of the organ.

The old term "surgical kidney" was applied to all secondary purulent lesions of the organ resulting from primary septic foci in the lower urinary tract. The name pointed to the origin of the renal lesion. But suppuration in the kidney may occur "spontaneously" as well as a result of vesical infection. Much light has been thrown on this subject by Traube, Niemeyer, Lister, Roux, Albarran, Klebs, Lepine, Clado, Escherich, Rovsing, Morell, Guyon, Melchior, Reymond, Horton Smith and Pernice, and Scagliosi. The light generated in the laboratory of the scientific inquirer is now guiding the surgeon in his every-day routine methods, and is enabling him to safely navigate his patient through what were not long ago hidden perils, showing him how to employ practical safeguards against dangers which may threaten the life of his patient.

It is now agreed that renal suppuration is the direct result of the invasion of microbes from some source or other, or of the action of their toxins. The organisms which induce

suppuration in the kidney differ in no respect from those which produce sepsis in other tissues, but it is rather remarkable that, long after the importance of asepsis was recognised in connection with the surgery of wounds, the greatest carelessness was displayed in the use of instruments introduced into the bladder, and in operations upon that viscus. Special names were even employed for septic infection of these parts, such as "urethral fever," "urinary fever," "catheter fever," terms which entirely obscured the true nature of the maladies, and the use of them now is to be condemned.

A study of the micro-organisms found in the urine, and their significance in diseases of the kidney, is of great importance to the surgeon. The value of a careful bacteriological examination of the urine has not until lately been sufficiently appreciated, but as surgeons come to be better acquainted with the methods of bacteriological research they will be more willing to employ them as an aid to diagnosis. For our present purpose it is necessary only to consider the methods employed in examining the urine for micro-organisms giving rise to septic conditions pure and simple, and leaving out of account such microbes as the tubercle bacillus, which is not truly a septic organism, although its presence materially predisposes to septic invasion.

Normally the urethra is found to be crowded by micro-organisms, only some of which, however, are pathogenic; indeed, the mucous membrane and the mucous glands are so infested with microbes that even prolonged and what may seem very thorough cleansing with mild antiseptic solutions fails to sterilise the passage. Even under normal circumstances this condition prevails as far up as the sphincter, but within it the urine is sterile. In health the kidney tissue is free from micro-organisms, and, even when they are introduced into the organ, the renal tissue appears to have a remarkable germicidal power and a capacity of eliminating them and their toxins from the system. To prevent contamination in the passage of the urine through the lower urinary tract certain precautions are necessary. It may be accepted that the healthy urine as it is excreted is sterile and the passage through which it flows, with the exception of the lower part of the urethra, is free

from organisms, but while this is so certain precautions must be taken to ensure that the urine is not contaminated prior to or during its collection. In a healthy person, if the urethra of the subject and the hands of the examiner are carefully cleansed and sterilised, the urine drawn off by a sterile catheter, conveyed into a clean vessel, and shut off from contamination, decomposition does not take place, and no micro-organisms are found even although the urine is kept for a long time.

Another point of importance is that the urine gives very early evidence of the presence of micro-organisms in the blood; they may be excreted by the kidney without causing any change in the organ, and without altering the ordinary physical characters of the urine. The urine may be clear and acid in reaction, but usually it contains a trace of albumen, and in most septic diseases it may be shown by culture experiments to contain bacteria, consequently micro-organisms in the urine do not by their presence necessarily indicate disease of the urinary tract.

The sterile character of healthy urine has long been known—it was first demonstrated by Lister and by Pasteur many years ago—but although this has been known and taught, the full significance and importance of infection of the urinary tract has been until lately almost universally disregarded. This neglect has caused much harm and confusion, and has caused many theories to be brought forward to explain symptoms which are now known to be due to septic poisoning.

The first difficulty which presents itself in a bacteriological examination is to obtain the urine free from external contamination. To make a reliable observation the urine must be conveyed from the bladder to a sterilised vessel, all risk of contamination being obviated. How then are we to proceed? The appliances, namely Melchior's catheter, tubing, and collecting vessels must be sterilised by boiling in an alkaline solution, and afterwards washed in sterilised water; the glans penis and the prepuce, but especially the meatus, must be thoroughly washed with soap and water containing a little ammonia; this is followed by washing with spirits of wine, and afterwards with carbolic acid solution 1 in 40 in water. The meatus

should also be douched with sterilised water, afterwards the mucous membrane should be carefully everted and again thoroughly cleansed. All the parts of the patient having been thoroughly disinfected, the surgeon again cleanses his hands, and lubricates the catheter with glycerine containing carbolic acid 1 part in 40, and passes it into the bladder. The urine is allowed to flow direct into the tubes of the centrifugal separator, and after a few minutes in the machine any deposit of microbes is thrown down, and can be at once examined microscopically, or by culture observations. The advantages of this rapid method are obvious.

The organisms which invade the urinary tract may be divided into two classes—those which induce decomposition of the organic constituents of the urine, and those which cause little or no chemical change in these substances. This distinction is a very important one. When organisms which do not possess the power of decomposing the organic constituents of the urine are introduced into the healthy urinary tract they fail to produce any serious effect. For example, a pure culture of the bacillus coli or of the tubercle bacillus may fail to induce cystitis when injected into the uninjured bladder, but if along with them urea-decomposing organisms are introduced, acute cystitis is sure to follow. The principal organisms which induce decomposition are the staphylococcus pyogenes aureus, and albus, some staff bacilli, and diplococci. The latter may be non-pyogenic or pyogenic.

In the latter division are placed the streptococcus pyogenes, the coli bacilli, and the tubercle bacilli.

Excluding cases of catarrh of the kidney where the irritation is the result of the action of chemical agents, or of mechanical irritants such as renal calculi, inflammation is the result of pyogenic micro-organisms. This fact has been established as the result of many observations. While aseptic chemical and mechanical irritants may produce the formation of small quantities of pus, the pus so formed is free from organisms, and when absorbed does not produce secondary abscesses. But while this statement must be accepted, it is also obvious that the resistance of the part subjected to such continuous irritation must be seriously diminished. Consequently an injured

kidney, or a kidney with a stone in it, is very liable to become septic.

The organisms which produce morbid changes in the kidney do not differ from those found in other tissues; indeed, almost all the microbes which are pyogenic in man gain an entrance to the urinary tract, and have been discovered as causes of disease in these parts. The two most frequent sources of infection are through the intestine on the one hand, and *via* the urethra on the other. In the former the organisms reach the kidney through the blood, in the latter along the urinary tract, or through the lymphatics.

The bacillus coli communis first recognised in 1886, by Escherich, is normally found in the intestine; but in diseases affecting the alimentary tract the number is enormously increased, and in certain well-known conditions the virulence of the organism is intensified.

Clado in 1887 isolated an organism which he named "bacterie aseptique de la vessie," and a year later Halle and Albarran described the same bacillus under the term "bacterie pyogene." In 1891 Morell identified these microbes with the bacillus lactis aërogenes of Escherich; the year following Krogius, and not long after Melchior, showed them to be identical with the bacillus coli communis. The question of names has therefore led to much confusion in the past, but now, fortunately, the subject is cleared up considerably by the work of Rovsing, Denys, and others.

In the urinary passages, as elsewhere, the virulence of the bacillus coli varies according to circumstances. In some cases its presence causes little or no disturbance, while in other instances its virulence is great, and death may take place even before suppuration is established, as in acute septic interstitial nephritis. The bacillus coli is by some observers supposed to be the most frequent cause of inflammation of the urinary tract, being in most instances absorbed into the circulation through lesions in the intestine and deposited in the kidney. This occurs most commonly when the intestinal tract is obviously diseased. It is found, as a matter of experience, that pyelitis or septic nephritis comes on very commonly in the course of convalescence from enteric fever, dysentery, or acute

diarrhoea. The bacillus coli or its products probably also plays an important part in the etiology of certain nervous lesions associated with chronic inflammatory diseases of the urinary tract. The exact pathological position of the bacillus coli has not been, however, satisfactorily determined, and that position is rendered more difficult to ascertain by post-mortem examinations, from the circumstance that it multiplies with great rapidity and penetrates even distant parts after the death of the individual, and may even spread from the intestine to the closed cavities, organs, and fluids of the body. Any injury of the intestine, or even injury of the tissues in its neighbourhood, seems to be sufficient to promote the emigration. Probably the microbes are transuded from the intestine very much in the same manner, and through similar causes as induce the exudation of serous fluid; in fact the two transudations are often coincident, but their relationship to one another is not fully understood. Probably the serous exudation is a result of the migration of the microbes.

The bacillus coli communis is a short rod resembling the bacillus of enteric fever, but it has rounded ends and possesses the power of movement due to cilia, which are often double or multiple, but sometimes single; it is easily isolated. On agar, within 24 hours large superficial and small deep colonies form; these are denser and more glancing than the enteric bacillus, and of a slightly brownish-white colour. On potato the growth is also of a brownish tinge, spreads rapidly, and bubbles of gas are seen around the colonies. In this respect it contrasts with the enteric bacillus, the colonies of which are colourless and free from gas.

In the intestine it is clearly non-pathogenic, but when it migrates to other parts, whether living or dead, it is strongly pyogenic. The soluble products of the growth of the bacillus seem to be the same in their action whatever may be the medium employed, but the source from which the organisms is derived has a very important influence on their action. It is a fact worthy of note that the organisms taken as a pure culture from a peritonitis arising from a perforation of the intestine are more virulent than when taken from a healthy intestine; or again, if the intestine be obstructed artificially

the activity of the bacillus is also increased. In different cases of pyelitis and cystitis the same peculiarities may be observed ; but while the organisms derived from one case on cultivation and inoculation into animals may produce very mild effects, those taken from another case which appears to be exactly similar may induce very grave results. The bacillus coli seems also to have the power of increasing the activity of other bacilli, or of rendering their product more harmful.

In the normal state micro-organisms fail to penetrate within the sphincter vesicae, and while they may gain access from above they may be voided without producing any lesion in the urinary tract. Observations, clinical and experimental, show that microbes undoubtedly play a most important rôle in sepsis of the kidney and the urinary tract, but while this is so there are certain conditions necessary prior to the micro-organisms establishing their footing in the organ. With increased knowledge of the life history, and of the conditions necessary to the propagation of bacteria, the infection by microbes becomes an increasingly complex phenomenon. At one time it was considered that all that was necessary to induce an invasion of microbes was the introduction of pathogenic organisms into the system, but it is now known that many conditions and circumstances require to be attended to, both in the cultivation of germs outside of the body and in the inoculation experiments upon animals, otherwise the parasites fail to propagate. In cultivation experiments, for example, the circumstances affecting their growth are numerous—the nature of the soil—the pabulum upon which one organism thrives may prove quite unsuitable for another, or, again, the medium may be appropriate, but the vigorous growth of the microbe may be retarded by such influences as electricity, light, temperature, the presence or absence of certain gases, etc. Then, in the successful inoculation of animals there is the whole question of susceptibility and immunity, natural or acquired ; phagocytosis, and the bactericidal influence of the body fluids. These all play important parts, so much so that we do not know altogether what forms an open door in one case and an effectual barrier in others ; but we do know that the readiness with which pus is formed depends upon

conditions belonging to the host as well as upon the characters of the parasite.

In the healthy individual there are natural barriers to the invasion of microbes to which we must call attention, and in doing so we shall refer to the mucous surfaces only—the air-passages, the alimentary canal, and the lower urinary tract. These are the natural portals of infection. Take first the air-passages. The circumstance that the air occupying the smaller air-passages is free from septic germs was first pointed out by Lister. This fact he explained by the theory that one of the functions of the air-passages is to filter out the dust and germs. It is also known that haemorrhagic infarctions of the lung may remain sterile for long periods. The subject of the resistance of the air-passages to the invasion of microbes has been worked out carefully by Gunning, Strauss and Dubreuil, Sinclair Thomson and R. T. Hewlitt, Würtz and Lermoyez, and others. Those who have inquired into the matter believe that in a healthy animal the mucus, and the air contained in the trachea are free from septic micro-organisms, and some observers hold that the nasal mucus itself exerts a bactericidal influence. It must be remembered, however, that true blood-parasites, such as those of anthrax, chicken cholera, the spirilla of relapsing fever, and the plasmodia of malaria, may infect the host through the lungs. Take now the alimentary canal. The experiments of Strauss and Würtz and many others show that strong germicidal power is possessed by the healthy gastric juice, especially when it contains free hydrochloric acid. It quickly destroys most germs, amongst which may be mentioned the cholera spirillum and the typhoid bacillus as well as the bacillus pyocyaneus, while the pus cocci show greater resisting power. The spores of most micro-organisms resist the action of the gastric juice, or are more difficult to destroy than the organisms themselves. Pasteur and Toussaint showed that even in anthrax the healthy mucous membrane of the stomach and intestinal tract offered effective resistance, but that when the mucous membrane was injured, invasion was almost certain to occur. Similarly, Orloff proved the protective power of the mucous membrane against staphylococcus pyogenes aureus. He fed animals for three months upon infected food and failed

to produce any evil results *so long as the mucous membrane* of the alimentary tract *was intact*, but when the mucous membrane was injured artificially the organisms present in the alimentary canal became absorbed, and formed infective emboli in distant parts.

This leads us to the consideration of the conditions of the resisting power of the mucous membrane of the bladder. About the earliest experiments were those of Fels and Ritter. They produced ammoniacal urine and cystitis in dogs by inoculating their bladders after ligaturing the urethra, but they found that as soon as the ligature was removed the bladder resumed its normal condition. Guyon, in 1888, proved that pure cultures of various pathogenic micro-organisms might be introduced into the healthy bladder without inducing a cystitis, and since then other observers have carried out extensive researches to show that the presence of pathogenic bacteria in the healthy bladder does not necessarily induce cystitis. The writer has shown that tubercle bacilli may be carried by the blood from distant parts and eliminated by the kidney without any morbid change in structure being induced in the urinary tract ("Tuberculous Disease of the Kidney," *Lancet*, February 24th, 1900, *et seq.*) Pernice and Scagliosi (*Riforma Medica*, No. 98) injected pyogenic micrococci into the blood of animals, and in many instances found them afterwards in the urine without any septic lesion having been induced either in the kidneys or the bladder. But when the resisting power of the mucous membrane is diminished, by the presence of a foreign body, by reason of injury, or as a result of the presence of residual urine, then infection is liable to take place. Reymond (*Ann. des Mal. des Organes Génito-urinaires*, p. 253, 1893) clearly demonstrated that in order that active inflammatory changes may follow the injection of microbes into the bladder the mucous membrane of the organ must be damaged in some way, or retention of urine must be induced. Strauss and Germont, on the other hand, showed that simple mechanical obstruction by aseptic ligature of the ureter does not induce any changes other than atrophy of the corresponding kidney; whereas, according to Charcot and Gombault, septic ligature of the ureter produces septic pyelitis. In the

same way injury produced by blows or falls may act as a predisposing cause in preparing the soil for septic invasion. This was recognised by ancient writers as a clinical fact, but in order to demonstrate the fact experimentally Albarran contused one kidney in a rabbit and injected a pure culture of a pyogenic organism into the animal's ear. The injured kidney showed evidence of commencing suppuration the day following.

Another point of importance is one which must be recognised by every clinical observer, viz.: that without the introduction of micro-organisms, retention of urine does not of itself give rise to inflammation. According to the observations of Aschoff, Schmidt, and Schnitzler, the *bacillus coli communis* is most commonly present in the urine in cases of cystitis, also the *bacillus proteus vulgaris*, and not infrequently pure cultures of these organisms may be obtained from the urine. The etiological relationship between the organisms and the inflammatory changes has been clearly demonstrated, but more information is required respecting the precise conditions which regulate the virulence of septic attacks, especially those resulting from lymphatic invasion.

An important observation bearing upon the etiology of sepsis of the bladder is that in the large majority of cases the viscus had been examined either by the finger or by instruments prior to the onset of cystitis.

As the result of experimental inquiry, it may be asserted:

1. That simple retention of the urine does not give rise to septic inflammation.

2. That small cultures of pyogenic micro-organisms, such as *staphylococcus pyogenes aureus*, *staphylococcus pyogenes albus*, or *bacillus coli communis*, when introduced into a healthy bladder fail to produce sepsis.

3. That if the mucous membrane of the bladder be injured or diseased prior to the introduction of micro-organisms, sepsis immediately occurs.

4. That if artificial retention of urine is induced from six to twenty hours after the introduction of the septic organisms into the bladder, suppurative inflammation of the mucous membrane follows. The bearing of these observations does not require to be pointed out at present.

We may now consider the *conditions which tend to prevent, and those which facilitate, septic inflammation of the kidneys.*

While the suppurative diseases of the kidney form a goodly proportion of the cases which come under the care of the surgeon, if looked upon from a wider standpoint the observer is at once struck with the comparative rareness of suppurative lesions, as contrasted with the non-suppurative maladies of the kidney, and also with the fact that lesions of that organ characterised by considerable accumulations of organic fluid, or by the presence of masses of foreign matter, may remain sterile for long periods.

The anatomical situation of the kidney protects it to a considerable extent both from injury and from infection; its deep situation in the lumbar region, far away from air contamination; its independent lymphatic supply, which does not communicate, except in the most restricted way, with the surrounding tissues, but only with that of the urinary tract, also tend to isolate the kidney and protect it. It is also shut off from the abdominal cavity, and is surrounded by a firm fibrous capsule through which very few blood-vessels, and almost no lymphatics, pass. In this way it is protected from invasion by microbes except by certain channels which will be referred to presently. Over and above these conditions, we have the power of the bladder to resist, within certain limits, the propagation of micro-organisms, and intercept their access to the kidneys. We have also the important physiological action of the ureters, the guardians of the kidneys, from which the escape of urine is intermittent and not continuous, and, at the same time, urine is permitted to pass in a downward direction, while an efficient barrier is provided to prevent a backward flow. The power of the bladder to completely empty itself at regular intervals, and so wash away any contamination, is also an important safeguard. In youth and early manhood moderate care in the use of instruments and due regard to cleanliness almost never induce any evil results. In cases of retention of urine from spasmodic stricture, alcoholic coma, or in the retention of specific fever, etc., cystitis or ascending pyelitis is seldom seen; the

mucous membrane and the muscular wall of the bladder are healthy in such cases, and the viscus is soon capable of completely emptying itself. These may be regarded as the physiological protectors of the kidney, which must be overcome, in one way or another, before septic inflammation can be induced.

The factors which facilitate an invasion of microbes are any circumstances interfering with these physiological conditions. In the decline of life the picture is entirely changed from what is seen in the earlier stages of manhood, the whole vital energy of the body is diminished, and the generative power of cells becomes enfeebled, the tissues which are rendered effete by their own functional activity are no longer replaced by others in their earlier completeness. The nervo-muscular energy becomes defective, the bladder loses its expulsive power; between the muscular fasciculi there is a new formation of connective tissue which is at first soft and cellular, the recurring pressure during micturition causes it to yield between the muscular bundles carrying the mucous membrane with it; through time cirrhosis sets in with atrophy of the muscular elements, till ultimately a number of interlacing bands give to the internal surface of the bladder somewhat the appearance of the trabeculae of the ventricles of the heart. The bladder is now unable to empty itself completely, gradually the orifices of the ureters dilate, the flow from the kidneys is in a slow continuous stream, and the column of fluid, in place of being interrupted, is unbroken from the neck of the bladder upwards to the renal pelves. If the smallest inoculation be now made even into a bladder which previously contained clear urine only, rapid sepsis is certain to follow, and involve not only the bladder but the kidneys also. The physiological barriers have been removed, and if once the invading army of microbes gain a footing on the soil nothing will drive them back. In such cases the utmost skill and care is required in the treatment of the patient. The soil is rich and ready for the seed—the surgeon must endeavour not to plant it, although it is around him on all sides. He may often succeed, but in a few instances, even when employing all the aseptic precautions, and using in addition antiseptic solutions, the cleansed catheter

may detach pyogenic organisms from the folds of the urethra, and push them onward into the bladder.

If inquiry be made respecting the disease antecedent to pyelonephritis, it will be found that lesions producing obstruction to the flow of urine from the bladder are the most frequent cause. Hence suppurative disease of the kidney may be looked upon in many cases as the natural termination of such conditions as stricture of the urethra, diseases involving enlargement of the prostate, cystitis, vesical calculus, loss of expulsive power of the bladder from atony, either as a consequence of grave nervous lesions or from local disease. These, however, are but the remote causes. Not only is it necessary that the urine be retained, but certain morbid changes must be produced in its composition. The excretion of the kidney when preserved from external contamination either inside the body as in hydronephrosis, or outside in a sterilised vessel, will keep for an unlimited period without undergoing any putrefactive change; but if inoculation of germs occurs, then the pent-up fluid rapidly undergoes decomposition. Hence the danger of instrumental manipulation in such cases.

Certain injuries and diseases of the general nervous system at once decrease the natural resisting power of the bladder. Even although no instruments have been used, septic contamination may rapidly occur when the control of the nerve centres is impaired or suddenly cut off. Again, the influence of the local nervous system upon inflammatory processes, either by impairing the nutritive activity of the part, or by diminishing phagocytosis, is most important, and it is difficult to say how far instrumentation of the urethra or the bladder acts by reflex irritation through the nervous system. The presence of an impacted stone in the urethra may lead to inflammation of the testicle, or the passage of a sterilised bougie may be followed by temporary suppression of urine, succeeded by renal haematuria for some days. The suppression, probably, is due to a reflex spasm of the renal arterioles. To cause complete suppression of urine, the spasm of the arterioles must be very great, and must involve the majority of the smaller vessels. The extent and gravity of the disturbance of

the circulation is clearly indicated by the abnormal constituents in the urine when the attack has passed. In such cases the circulation in the bladder is also interfered with, and should any contamination have been introduced, the danger of very acute sepsis (acute septic nephritis) of the whole urinary tract is great.

When the urinary organs are reasonably healthy throughout, and the bladder can completely empty itself, even unclean instruments introduced occasionally during long periods of time do not necessarily lead to a septic condition of the urine. I have known a carter carry a catheter in his pocket, and an engineer who extemporised a bougie from 18 inches of telegraph wire, and these instruments were used for years, when required by the individuals, without doing any perceptible harm. But, if the bladder fails to empty itself completely, as from urethral stricture, from local or general paralysis, or enlargement of the prostate, vesical calculus, tumours, or any other cause, then the danger of sepsis being induced by the most minute particle of infected matter cannot be exaggerated. The mucous membrane loses its resisting power, and the retained contents of the bladder become contaminated by even the smallest inoculation of infected material introduced from without. With the conditions mentioned, when once introduced, the organisms multiply and rapidly spread, causing chemical decomposition of the urine and structural alterations of the mucous membrane and walls of the bladder.

What is true of the conditions which induce sepsis of the bladder, also applies to the predisposing causes of septic invasion of the kidney.

We have now to consider *the modes of attack or the channels along which the invasion of the kidney by microbes may occur*; also, very briefly, the histological changes produced in the kidneys.

Septic lesions of the kidney may be induced by:

1. Infarctions arising from infective emboli conveyed through the blood-stream from tissues or organs foreign to the urinary apparatus.
2. Invasion along the lymphatics of the urinary system.
3. Contagion along the lumina of the excretory ducts.

4. Septic contamination by contiguity with abdominal organs.

5. Wounds.

6. Presence of foreign bodies.

It may here be remarked that while, primarily, the infection is conveyed to the kidney by one of these channels, as the disease advances the infective process becomes more complicated, and is a combination of some of those already mentioned.

1. *Invasion by the blood.*—In purulent embolic nephritis the virus is conveyed by the blood-stream from a primary septic or otherwise infected focus in tissues or organs foreign to the urinary apparatus, and corresponds to what has been described as descending infective nephritis. Long ago Cruveilhier, Reverdin, and Verneuil showed that in patients who died from anthrax the kidneys presented grave lesions, due to infective emboli, which we now know to be formed principally by the specific micro-organisms of the disease. In a similar manner acute suppurative disease of the kidney has been found to follow osteomyelitis, a disease which was first demonstrated by Pasteur, Klebs, and Max Schüller to be of an infective nature; and Ribbert and Rodel, by a carefully concluded series of inoculation experiments showed many years ago the wonderful rapidity with which multiple suppurating foci are developed in this disease. The primary lesion may be erysipelas, pyaemia, one of the eruptive fevers, malignant endocarditis, suppurative diseases of the skin, the lungs, or the alimentary tract, or of the serous surfaces; but whatever may be the original infective disease, the secondary foci in the kidney breed true. In pyaemia and in erysipelas the streptococcus has been found, and likewise in osteomyelitis the staphylococcus has been demonstrated as the exciting cause of the renal abscesses. But whatever may be the precise nature of the microbes, the course of events and the histological changes in the kidney are much the same. The lesion in the kidney is embolic, but there is more than simple obstruction to the circulation, the infection being derived from a part where putrefactive, inflammatory, or other changes due to the presence of microbes are going on. The embolus possesses infective properties, so that in addition to plugging of the renal arterioles, it sets up

inflammatory processes, both in the vessels within which it has become impacted and in the surrounding tissues. Immediately in the neighbourhood of the embolus the tissues show evidence of necrosis, and beyond this area the connective tissue softens and becomes infiltrated by leucocytes. The embolus is loaded with microbes, which, in the early stage of the process, may be seen in the clot, but very rapidly the infected fibrine breaks down and the organisms may speedily spread to more distant parts, and be found in the glomeruli and capillaries. The organs are enlarged and elastic. The capsule is easily separated, and between it and the cortex there may be numerous small haemorrhages, in each of which there is a small yellow spot. These are the miliary abscesses, with their surrounding hyperaemia. The abscesses project slightly from the surface of the kidney, as small round miliary prominences, some of a deep red colour, others white or yellow surrounded by a deep zone of injected vessels, and on account of the anatomical arrangement of the renal vessels the suppurating areas are more abundant in the cortex than elsewhere. The contents of the smaller abscesses are composed of disintegrated fatty glandular tissue, mixed with pus, and in the centre of these collections of inflammatory products colonies of micrococci are seen. These microscopic bodies only involve a glomerulus or a minute vessel, but at the same time the inflammatory process extends to the surrounding tissue, in a manner similar to what has been already described as the mode of extension of inflammation from the uriniferous tubules in cases of pyelonephritis. On examining a section made from the cortex to the pyramids, the inflammatory process will be seen to follow the distribution of the renal arterioles, and, when large in size, to assume the form of a cone with the base towards the periphery of the kidney. When a section is made of these larger abscesses, their contents escape and leave a ragged cavity. The contents are soft and pulpy, and on careful examination will be found to be impregnated with micrococci, while the walls will be observed to be composed of leucocytes and red blood corpuscles, which have infiltrated the renal tissue. Whether the infection be due to pyaemia, malignant endocarditis, or other infective disease, the pathology of the renal lesion is very much the

same. The process goes on to suppuration and destruction of the infected area. Both kidneys are generally affected, the abscesses are usually small and multiple, and, on account of the arrangement of the blood-vessels, the suppurating areas are more abundant in the cortex than elsewhere. There they are rounded, or in the form of cones with the bases towards the capsule. In the medulla they are more elongated, and follow the distribution of the vessels.

Infection by means of the blood-stream is not limited to the method just described. We also meet with instances of auto-infection of the kidney, or examples of the blood-vessels carrying infective material from one portion of the urinary tract to another. The earlier formed foci in the kidney may be due to an ascending infection from the lower urinary tract, while the later lesions are the result of minute infective infarctions. When the emboli are numerous the patient succumbs before the abscesses attain considerable size, the infection of the kidney being only a part of a widely-distributed septic poisoning. Septic kidney, following any of the acute diseases mentioned, may, however, become chronic by the abscess rupturing into the pelvis, but this only occurs when the infective infarctions are few in number. How frequently we find cases of pyuria, which we may attribute to pyelitis, dated back by the patient to the time when recovery was taking place from an attack of fever, of erysipelas, or some puerperal complication. In many such cases the sepsis in the kidney is doubtless embolic in origin, although it is difficult to demonstrate the fact. The history of these cases is sometimes so clear that it is impossible to resist the conclusion that the acute general disease is related to the local renal lesion as cause and effect. The connection between septic nephritis and the primary lesion has been carefully studied by a considerable number of investigators, amongst whom may be mentioned Davaine, Colin, Tedenat, Charrin, Barrette, Coze, Feltz, and Lindsay Steven.

CASE I.

Compound comminuted fracture of humerus and injury to elbow-joint, septic poisoning and suppression of urine (acute embolic nephritis). Metastatic abscesses in kidneys only.

An engineer, aged 28, got his arm crushed between two railway-carriage buffers on 1st November, 1891; the elbow-joint was opened, and there was a compound comminuted fracture of the lower end of the humerus. When seen a week after the accident the arm had been amputated in the middle third of the humerus, and the patient presented the symptoms of septic poisoning, together with complete suppression of urine during the previous twenty-one hours, and he died the following day. On post-mortem examination the following appearances were noted in the kidneys. Before removing the fibrous capsule a number of minute abscesses were seen as round, white, miliary prominences. These separated the capsule from the surface of the cortex, and when the capsule was removed the contents of these small abscesses escaped. On exposing the surface of the cortex, it was found to be well studded over with miliary abscesses, some of which were of a reddish brown colour; others, the smaller ones, were red at the circumference, and of a pale yellow colour or white in the centre, while the larger ones were pale throughout. On section these miliary foci were seen to be arranged in the shape of a cone with the base towards the periphery of the kidney, and recalled the appearance of the pyramids of Ferrein.

Under the microscope it is seen (Fig. I.) that the abscesses arose by obstruction of the arterioles. At (A) the arteriole is shown to be plugged with a mass containing micrococci. The organisms have destroyed and penetrated the wall of the vessel, induced a slight but feeble proliferation of leucocytes (B), and entered the lumen of the uriniferous tubules (C). Colonies were also found in the vessels of the glomeruli, which appeared as dark granular masses about twice their normal diameter.

An instance where metastatic abscesses found in the kidneys and elsewhere without any external wound is recorded in *Lectures on Surgical Diseases of the Kidney*, page 191:

"On the surface were seen numerous small elevations; these were minute abscesses into which haemorrhage had occurred. On section they were seen to exist in every part of the renal tissue, but especially in the cortical region. These deposits were present in great abundance in both kidneys, their size averaging that of a pin head. Similar deposits were found in almost every part of the body. On

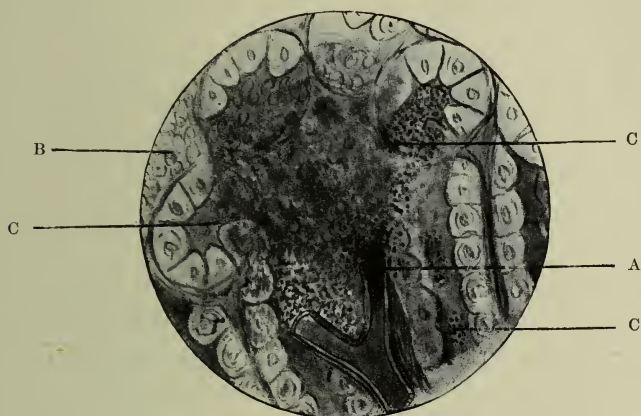


FIG. I.

the skin there were haemorrhagic spots, and the remains of what had been observed during life as an extensive crop of pustules; on the surface of the pia mater there were haemorrhagic and purulent spots; in the heart there was a softened patch about an inch in length and three-eighths in breadth; in both lungs numerous metastatic deposits, some haemorrhagic and some purulent; in the pleurae, soft lymph; in the spleen also metastatic deposits; and in the intestines similar deposits in remarkable abundance, appearing like berries in some of the blood-vessels. In the right knee there was some brownish fluid and a few minute abscesses in the muscles of the thigh, but no pus in the femoral vein.

“There was in this case no external wound detected, either before or after death, and the chief complaint during life was of the right knee.”

In place of originating a large number of minute and widely scattered abscesses by conveying small groups of bacilli, the blood-stream may carry an infected thrombus to the kidney and the vessel in which it becomes impacted is closed. The embolus may obstruct one large vessel and lead to a suppuration of considerable size, but limited to one organ. Mayer has reported in *Virchow's Archiv* a case in which a large embolus became impacted in the right renal artery and produced a large abscess, in the pus of which numerous bacteria were found.

Small branches of the renal artery may be blocked, and the septic lesion which is disseminated over the focus of distribution in the first instance may ultimately come to affect a much larger area.

This is probably one of the most common modes of infection where the lower urinary tract is not primarily involved.

2. *Invasion by the lymphatics.*—In order to appreciate the pathology of this mode of invasion, it may be necessary to make a few remarks respecting the lymphatic supply of the bladder, ureters, and kidneys. When an injection mass is forced into the ureter, the mucous membrane having been previously lacerated, the fluid proceeds directly into the lymphatic vessels surrounding the ureter, passes downwards towards the bladder, and upwards in the direction of the kidneys. In this way it may be shown that the course which the lymphatics pursue from the bladder is one of direct distribution along the submucous connective tissue surrounding the ureters, to the capsule of the kidney. They then penetrate the renal substance, and lie in spaces between the uriniferous tubules, so that when, in a kidney thus injected, a section is made of the cortex, the lymphatic vessels can be easily seen extending in thin streaks from the extreme cortex towards the medulla. These channels form a most intimate ramification in the parenchyma of the kidney, much more minute, indeed, than the vascular capillaries, as these vessels, as well as the

uriniferous tubules, are closely surrounded by lymphatic spaces.

In acute septic nephritis, without suppuration, the virus is conveyed from the lower urinary tract to the cortex of the kidney by the lymphatic channels. We meet with certain instances of very acute septic absorption resulting from injuries of the lower urinary tract, but especially of the upper portion of the urethra and the neck of the bladder, where a violent septic poisoning is induced without the mucous membrane of the ureters or the renal pelves being involved, or only implicated in the inflammatory process to a slight degree. In the most of these very acute cases the patient has suffered previously from chronic albuminuria and cystitis. Suddenly, soon after an operation upon the urethra or the bladder, he has a violent rigor and suppression of urine, which may continue, or be followed by a more or less profuse renal haematuria. The further development of the case is practically that of acute septic poisoning, with profound collapse, which may terminate fatally within forty-eight hours. It is, indeed, a form of so-called "acute urinary fever."

On examination of the kidneys, the pelves and the ureters are found to be deeply injected, but free from erosion or ulceration, the mucous surface being intact. The kidneys are enlarged, flabby, dark chocolate-coloured, and when the capsule is cut the cortex protrudes almost like splenic tissue. When cut into the cortex and medulla are found to be deeply injected, and the line of demarcation is indistinct; no suppurating points may be seen, but on microscopic examination the whole of the kidney is found to be completely permeated, and the walls of the bladder and the ureters are likewise infiltrated with septic micro-organisms, which may give a perfectly pure culture on bacteriological examination.

We have here represented the most acute form of septic absorption. The invasion by microbes through the lymphatics is so sudden, and sometimes so overwhelming, that the patient succumbs before even minute abscesses have time to form.

CASE II.

Enlarged prostate, retention of urine, acute septic poisoning by absorption through the lymphatics of the bladder inducing acute septic nephritis without suppuration.

A farmer, aged 73, had suffered for three months from difficulty in micturition, and, from what he said, it was evident that for a much longer period an enlargement of the prostate had prevented him from completely emptying his bladder. The urine was clear, acid in reaction, contained a moderate quantity of albumen, but no pus, blood, sugar, or crystalline deposit. He was usually easily relieved from any difficulty in micturition by a warm bath, but on the 27th October, 1897, after drinking rather heavily, he found early in the following morning that he was unable to pass water. He waited till after mid-day on the following day before seeking assistance. By this time the bladder was much distended and the patient was in agony. The orifice of the urethra and the parts around were carefully washed with soap and water and cleansed with carbolic solution, the urethra was washed out with boracic acid solution, and the catheter was sterilised immediately before using it. The greatest care was employed in the use of the catheter, the regular employment of which was necessary; the patient was kept in bed and confined to light diet. All went well till the morning of the 16th of November, when the patient took a sudden and violent rigor, with a rise in temperature from normal to 104.4° F. The pulse ranged from 125 to 135 per minute, but good in quality, respirations 22 per minute. The tongue was dry and the lips parched. The patient said that he had no unusual pain in the bladder or in the region of the kidneys, but that he felt very ill and suffered severely from headache. He was unable to take any food. The skin was moist and soft, no vomiting and no diarrhoea. The urine drawn off in the morning amounted to three ounces. It was dark sherry colour, acid, sp. gr. 1025, contained a moderate amount of albumen but no blood, and only a few leucocytes. But the deposit removed by a separator contained a large number of organisms, which on

cultivation proved to be a pure culture of *staphylococcus pyogenes aureus*.

Diarrhoea set in, and the patient vomited a quantity of dark-green fluid on two occasions during the day, and in the evening the temperature reached 105° F. The tongue was dry and brown, and the teeth and lips covered with sordes; the skin was dry and very pale in colour. Four ounces of urine similar in character to that of the morning was drawn off. During the following night the patient was delirious, but the delirium was not very profound, the diarrhoea continued, and on the morning of the 17th the skin presented a peculiar mottled appearance, resembling that seen as a result of post-mortem staining. On passing the catheter no urine escaped. The temperature fell to 102° F., the pulse became rapidly feeble, during the day the patient became unconscious and lapsed into profound coma, with complete suppression of urine. In the evening the temperature fell to 97° F., and the patient died 53 hours after the first rigor which marked the onset of the septic intoxication.

Only a limited post-mortem examination was permitted, but it showed enlarged prostate, hypertrophy of muscular coat of the bladder, septic excoriations and submucous haemorrhages, numerous minute septic foci in bladder and a few in both ureters. The urinary bladder contained only two ounces of dark-brown urine. The muscular walls of the viscus were thickened, soft, and flaccid, the internal surface was marked by rugae covered by smooth but deeply injected and swollen mucous membrane. There was no ulceration, but close to the neck of the bladder, and in front of a much enlarged middle lobe of the prostate, the mucous membrane was excoriated, and the site of numerous submucous haemorrhages.

On laying open the ureters, the lumen of which was normal, and pelves of the kidneys, the mucous membrane was observed to be thickened and oedematous, and at short intervals there were irregular patches of deeply injected membrane, with here and there small pinhead-sized haemorrhages and small pale spots which proved to be miliary abscesses, but there was no erosion of the surface. Microscopic examinations of sections showed numerous small colonies of microbes between the

deepest layer of the epithelium and the basement membrane and between the basement membrane and the fibrous layer. At some points the basement membrane was completely destroyed, and the organisms were seen in the flat and cylindrical epithelial cells lining the ureter, but they seldom penetrated as far as the more superficial layer of pavement epithelium. Both kidneys were enlarged and the capsules were very tense, so that when an incision was made the softened renal parenchyma of the cortex pointed through the cut. On removal of the fibrous capsule, which was comparatively free, the surface of the kidney presented a peculiar mottled appearance seldom seen. On a dark chocolate-coloured background numerous

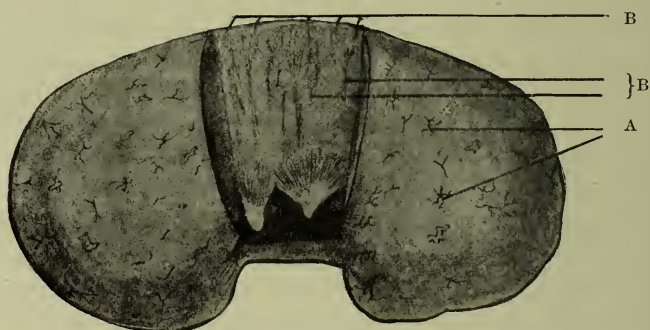


FIG. II.

still darker-coloured star-shaped and Y-shaped vessels were seen (Fig. II. A), almost like minute veins. These were not more than a fifteenth of an inch in diameter. On the surface the longest of these were about one-eighth of an inch long, but on section (Fig. II. B) they were found to extend almost as far as the line of separation between the medulla and cortex. The renal cortical tissue was very dark in colour and flabby, the Malpighian bodies could not be seen, but the line of separation between the medulla and cortex was distinct. Fresh inoculations from the renal parenchyma gave pure cultures of *staphylococcus pyogenes aureus*. A large number of sections made of the kidneys after hardening failed to reveal even microscopic points of suppuration, but throughout the substance of the organ, and especially in the extreme

cortex, numerous colonies of the organisms were found. These organisms were also found in the lymphatic spaces surrounding the ureters and in the walls of the bladder.

Fig. II. represents the left kidney; the aspect of the surface after the fibrous capsule was removed is shown, and the appearance of a perpendicular section through the middle third of the organ.

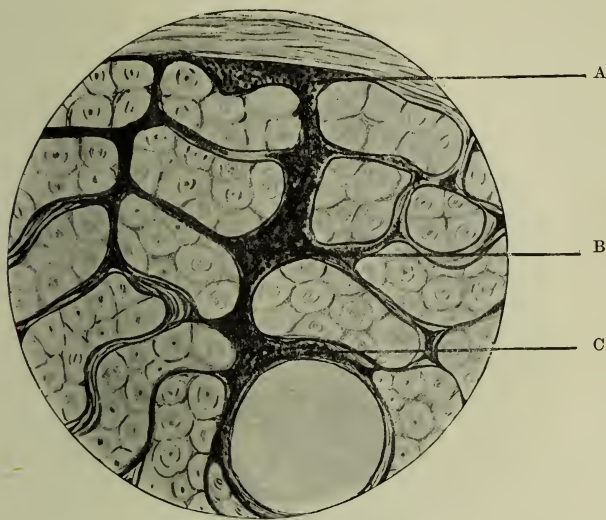


FIG. III.

Fig. III. is a microscopic section showing the colonies of organisms immediately under the capsule (A), and extending in the lymphatics between the uriniferous tubules (B) and around the Malpighian capsule (C).

Fig. IV. represents the microscopic appearances more highly magnified. The organisms are seen in the perivascular lymphatic spaces (A), in the lymphatic spaces between the uriniferous tubules (B), and in lymphatics around the Malpighian capsule (C), where the colonies have caused softening of the basement membrane and displacement of the Malpighian capsule. At (D) the organisms have migrated into the epithelium of the uriniferous tubules.

In many instances, however, the infection is not so violent.

The line of invasion can be more easily and clearly traced, and the time which elapses is sufficient for the formation of small suppurative foci. Here we have a form of *purulent interstitial nephritis*. The primary septic focus is generally in the bladder, and on laying open the ureter its walls are found to be thickened and infiltrated; but there may be no erosion of the mucous membrane, either of the pelves or of the ureters, as the presence or absence of ulceration depends upon the acuteness of the affection. The kidney is slightly enlarged,

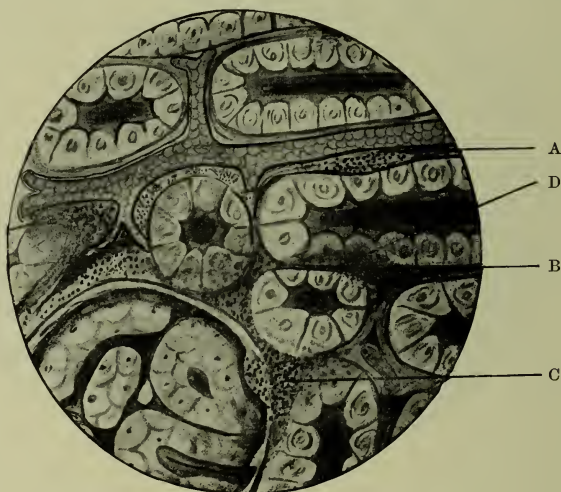


FIG. IV.

soft, pale in colour, the surface is smooth, the capsule is easily separated, and immediately under it a number of small pale bodies are seen, the majority of which are not larger than mustard seeds. On section the cortex and the medulla are seen to retain their normal proportions, and the line of separation is tolerably distinct. On microscopic examination the mucous membrane of the bladder presents the characteristic appearances of septic cystitis. The serous surface does not exhibit anything worthy of note, but on the mucous surfaces there are distinct areas of acute inflammation, the inflammatory products containing large numbers of micro-organisms. A section of the ureter shows the mucous membrane to be

normal or little altered in appearance, but the deeper structures are infiltrated with round cells containing micro-organisms. On examination of the kidney the small pale bodies are found to be minute abscesses, evidently of very recent origin; they do not follow the direction of the blood-vessels, but they are most abundant underneath the capsule, and although some of them are found in the cortex close to the medulla in this region they are less abundant. These abscesses are seldom found in the pyramidal region.

3. *Septic infection along the lumina of the excretory ducts* is the most common cause of pyelitis, pyonephrosis and pyelonephritis; but while the infective material is generally conveyed along the lumina of the ureter, pelvis, and uriniferous tubules in the first instance, in almost all cases the later lesions are produced by an auto-inoculation along the lymphatics or the blood-vessels. Suppurative disease of the kidney may be looked upon in many cases as the natural termination of such conditions as stricture of the urethra, diseases involving enlargement of the prostate, cystitis, vesical calculus, loss of expulsive power of the bladder through atony, either as a consequence of grave nervous lesions or from local disease. These are, however, but the remote causes. Then, again, undoubtedly, although not commonly, pyelitis, pyonephrosis, or pyelonephritis may be secondary to a limited purulent interstitial nephritis, and the renal suppuration may continue long after the primary disease has been recovered from.

When the flow from the ureters into the bladder is free, and the muscular action of the ureters is normal, inflammatory changes generally remain limited to the bladder, or only extend upwards by the lymphatics, but if the ureters are dilated, and the fluid columns between the bladder and the renal pelves are thereby rendered continuous, the bacterial invasion of the kidneys may take place through the columns of urine, the alkaline reaction of which greatly facilitates bacterial proliferation. In many cases the whole route of invasion may be easily traced by the morbid changes in the walls of the bladder, ureters, and pelves, right into the uriniferous tubules. The important distinction between this mode of invasion and that by means of the lymphatics is that, while

in the latter the abscesses are principally found in the cortex of the kidney, in the former it is the tubules of the pyramids which are the centres of infection. On microscopic examination of a case of pyelonephritis the tubules of the pyramids will be found to be blocked by elongated colonies of septic organisms, around which there is more or less intense inflammation, sometimes amounting only to a slight infiltration of leucocytes between the tubules, while in other areas the inflammatory changes are so advanced as to have caused complete disintegration of the renal tissue, which has come to be occupied by purulent cavities. Another point worthy of note is that in such cases micro-organisms are not, in the early stage of the disease at least, to be found in the blood-vessels, although, later on, septic thrombosis may occur. The microbes travel along the surface and interstices of the mucous membrane of the ureter till they reach the pelvis of the kidney; they then gain access to the uriniferous tubules, and in their lumina spread and multiply vigorously and give rise to multiple abscesses. This is not due necessarily to any obstruction to the escape of urine *per se*, but rather to the opportunity of infection by a septic focus in the bladder or elsewhere. When the pathogenic micro-organisms enter the uriniferous tubules from the pelvis, they form colonies and obstruct the lumina of the tubules, impair the vitality of the epithelium and basement membrane, which ultimately ruptures and allows the inflammatory products to pass into the inter-tubular lymphatic spaces, where they rapidly excite diffuse inflammation.

CASE III.

Old-standing stricture of the urethra, enlarged prostate, hypertrophy of the muscular wall of the bladder. Double pyonephrosis and pyelonephritis.

H. W., aged 79, was admitted to the Glasgow Royal Infirmary on the 6th September, 1898. Patient stated that until three weeks prior to admission he had not been troubled with his urine, but this statement is probably incorrect. However, it appears that about that time he had complete

retention, and called in a medical man, who relieved him by passing a gum elastic catheter, and this treatment was continued till admission.

When brought into the hospital the patient was in a very weak condition and in great distress. He complained of difficulty in micturition, severe pain in the hypogastrium and in the renal regions when he went for any length of time without passing water. A No. 8 catheter was easily passed, and when the urine was drawn off it was found to contain a large quantity of pus and blood, but no tube casts or crystalline deposit. The reaction was alkaline, and on cultivation *bacillus coli communis*, *staphylococcus pyogenes albus* and *staphylococcus pyogenes aureus* were found. The symptoms, beyond those referable to the urinary organs, were those of subacute septic poisoning. Considering the age of the patient, the thoracic organs were normal. Under treatment the condition of the urine improved greatly, and the suffering of the patient was relieved, but gradually he became weaker, and died on the 13th September. Only a partial post-mortem examination was allowed, and showed the following condition of the urinary organs :

There was evidence of an old stricture in the membranous portion of the urethra, which was dilated sufficiently to admit a No. 10 bougie, but the walls of the canal were hard and thickened ; the mucous membrane showed evidence of chronic inflammation. The passage on the proximal side of the stricture was considerably dilated, and the sphincter of the bladder was unusually large. The muscular wall of the bladder was greatly hypertrophied and the trabeculae were unusually prominent. The bladder wall was also slightly sacculated ; the mucous membrane was thickened, of a dark slate colour mottled with dark red patches, and here and there small erosions were seen, surrounded by small incrustations of alkaline phosphates. The ureters and the pelves were dilated, their mucous membrane swollen and deeply pigmented, the surface irregular and ulcerated, and at some points incrustated with phosphates deposited from the alkaline urine. The kidneys were enlarged and dark in colour, and the fibrous capsules were adherent and slightly thickened. The surface of the

organs after removal of the capsules was of a uniform dark reddish brown colour, soft and friable. On section the cortex was of a uniform dark colour, but close to the line of limitation the parenchyma presented a streaked appearance, pale lines being intermixed with dark red ones. These striae radiated in the form of cones the apices of which were towards the pelvis, and the bases did not extend into the cortex as far as could be seen with the unaided eye or with a hand-glass.

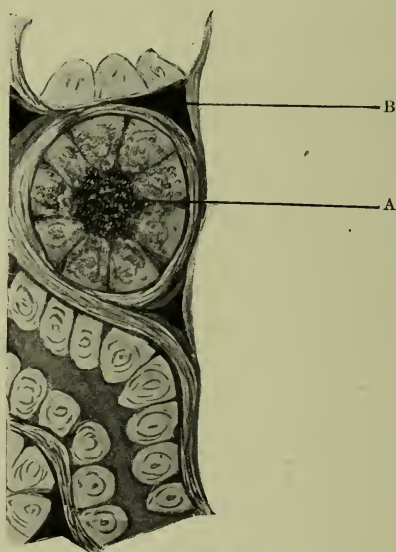


FIG. V.

On microscopic examination the tubules of the medulla were found in many instances completely blocked by bacteria (Fig. V. A), but their propagation in the kidney tissue was very irregular. Between the uriniferous tubules of the pyramids there were lines of organisms occupying the lymphatic spaces (B), and at other parts groups of leucocytes, some of which had degenerated and formed small abscesses; others presented the appearance of a very acute interstitial nephritis. The blood-vessels were deeply injected, while the epithelium of the tubules was swollen, granular, and in some places desquamated.

In this case the infection was a mixed one, but the course of events is very evident, both from the history of the case and the morbid appearances. The pathogenic and urea-decomposing organisms gained access to a bladder which had been previously over distended and habitually contained residual urine. From the bladder they extended upwards through the dilated and constantly patent ureters; they reached the pelves, where they rapidly set up a suppurative pyelitis, and there settled and multiplied vigorously. Producing small erosions at the apices of the pyramids, the organisms gained an entrance into the uriniferous tubules and into the inter-tubular lymphatic spaces, and multiplying in these situations they produced abscesses in the medulla. Only a few purulent centres were found in the cortex.

4. *Septic contamination by contiguity* is not common, on account of the isolated position of the kidneys, but under this heading we may place some of the abscesses of the kidney secondary to perinephric abscess, to psoas abscess, to abscesses of the liver or spleen, or to tubercular vertebrae. Again, in those instances where abscess of the kidney follows a contusion, fall, or a rupture of the kidney without a wound, may not the juxtaposition of the injured part to the colon facilitate contamination with the bacillus coli communis? Where severe injury is inflicted upon the organs situated in the lumbar region, the colon and the kidneys together, one can easily understand how infection may take place, even although there is no direct communication between the bowel and the injured tissues outside of it. The circumstance that rupture of the kidney is frequently followed by the formation of large abscesses, without any infection through the lower urinary tract or by means of the blood stream, proves almost conclusively that contamination by contiguity may occur. Experiments have demonstrated (Posner and Lewin) that the bacillus coli may easily pass through the intestinal wall when intestinal obstruction is induced, and these organisms may be found in the blood, and even in the urine. In cases of acute intestinal obstruction in man, I have found it in the urine along with blood, and when the obstruction was relieved the organism disappeared from the urine. I have also observed one or two

cases where an old hydronephrosis has become infected from the colon without any direct communication.

Reblaub also has recorded instances where pyelonephritis has been induced during the course of pregnancy, and the explanation he gives is probably correct, namely, that the pressure of the uterus caused a slight transitory hydronephrosis. Then some intestinal trouble intervening enabled the bacillus coli to enter the circulation, and flowing through the kidney, to find a suitable nidus in the pent-up urine. It is believed by some observers that residual urine in the bladder may be contaminated from the rectum.

5. *Wounds and injuries* of the kidney need only be mentioned as a cause of septic trouble.

6. *The presence of a foreign body*, such as a fragment of diseased bone ulcerating into the pelvis or the substance of the kidney, the presence of a calculus or a tumour, may act as a predisposing cause to sepsis. It is very seldom that a foreign body introduced into the bladder makes its way up the ureter to the kidney, and there setting up suppurative inflammatory changes gives origin to the formation of a calculus. The following case is therefore of special interest. A specimen was placed in the Western Infirmary museum by Dr. J. Lindsay Steven, who showed it at the Glasgow Pathological and Clinical Society at their meeting, 8th January, 1884. It was obtained from the body of a man who died from injury to the head, with laceration of the brain, etc. The patient died soon after admission, so that nothing is known as to the history of the renal condition during life. The suppuration of the kidney was due to a very uncommon cause, namely, the impaction of a foreign body in the pelvis. "As you will observe, the substance of the right kidney, except at its lowermost part, is occupied by large cavities, which, in the recent state, were filled with creamy pus. Lying in the long diameter of the pelvis of the organ there is a thick coarse bristle, to the lower extremity of which a branched calculus, composed of phosphate of lime, is attached. The ureter is slightly dilated, and the calculus extends into one of the calices. The bladder was found to be normal, but contained after death a small quantity of purulent urine. Left kidney normal."

PENETRATING WOUNDS OF THE EYE, COMPLICATED BY THE PRESENCE OF A FOREIGN BODY IN THE EYEBALL.

By A. MAITLAND RAMSAY, M.D., F.F.P.S.G.,
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PENETRATING wounds of the eye are at all times serious, and the prognosis becomes very much more grave when such injuries are complicated by the presence of a foreign body in the interior of the eyeball. In every large industrial centre such accidents are of daily occurrence, and few cases give the practitioner greater cause for concern, inasmuch as no matter how trivial an eye injury may seem, there is always the risk of disastrous after-results. The fate of the damaged eye depends indeed, in most instances, on the treatment first adopted, and at all times one's sense of responsibility is intensified by the knowledge that a wound of one eye may be followed by sympathetic inflammation of its fellow, and that this may be so severe as to cause complete loss of vision. The seven cases which follow have been recorded and arranged so as to present a clinical picture of the results, immediate and remote, that may follow penetrating wounds of the eye complicated by the presence of a foreign body in the eyeball.

1. W. T., aged 26, an ironturner by occupation, was sent to consult me in March, 1900, by Dr. Brown of Pollokshields. On the 2nd of October, 1899, he was struck on the left eye by a chip of steel, which perforated the cornea and embedded itself in the lens. The accident had apparently been followed by very little inflammatory reaction, and, when I first saw the patient, the eye presented a perfectly natural appearance, and

the visual acuity was quite up to the normal standard. When the pupil was dilated by atropine, and the eye examined by the ophthalmoscope, a minute black glistening particle was seen lying in the lens, which, however, with the exception of the area immediately enveloping the foreign body, had lost none of its transparency. Since a small foreign body situated in the lens does not usually cause inflammation of the eye, and as any attempt to extract this tiny fragment of metal would certainly have been followed by traumatic cataract, the patient was advised to let well alone, but to report himself regularly so that the eye might be kept under observation. For eleven months his progress was all that could be desired, but on the evening of the 26th January, 1901, the eye, without any warning, and for no reason known to the patient, became excessively painful, and sight disappeared completely a few hours afterwards. On the following morning the eye had lost perception of light, and was red, painful, and tender to touch, but of normal tension, the iris was discoloured, the pupil was contracted and did not dilate after the instillation of atropine, and the fundus oculi could not be illuminated. The patient was sent to bed and after treatment by atropine, opiates, leeches, and fomentations, the cyclitis subsided and light perception returned; but the media were so muddy that the interior of the eyeball could not be examined, and the iris, which was formerly of a light blue colour, had become a deep reddish brown. An X-ray photograph was taken, and demonstrated the presence of the foreign body (Plate 1, fig. 1) in the lens, exactly as it was before the occurrence of the inflammatory attack. As a result of the cyclitis the lens had become much more cataractous, and in consequence, on the 13th of March, it was extracted by a superior linear section. This led to great improvement of vision, and a skiagram taken ten days after the operation showed that the foreign body was gone. This case is still under treatment, and although the patient is now able to distinguish large objects there is great risk that the cyclitis may return and that the eye may ultimately require to be enucleated.

2. It is only very occasionally, however, that a foreign body is thus visible; in most instances it rapidly becomes concealed

by haemorrhage, or hidden by increasing opacity of the lens. It is therefore of the first importance that the eye be thoroughly examined at the earliest possible moment after the accident; and the nature of the latter, along with its attendant circumstances, ought always to be carefully and fully elicited. Obviously an eye which has been penetrated by the prong of a fork, or cut by a knife, is not at all likely to contain a foreign body; but, on the other hand, when the accident has been due to a blow from a chip of steel, of glass, or of stone, or where it has been the result of an exploding cartridge, or of a shot pellet, then the risk that the missile has become lodged in the globe is exceedingly great. The illustration (Plate 2, fig. 1) is from the eye of a girl, K. S., 17 years of age, who was sent to me by Dr. Newman, with the history that while she was working at a pithead on the 16th of February last she accidentally struck a dynamite cartridge, which exploded, blew off a part of her left hand, and seriously injured the eye on the same side. There was a small penetrating wound at the upper and outer aspect of the cornea, the iris was torn, the aqueous was stained by blood, and the lens was cataractous. There was great congestion of the circumcorneal blood vessels, much tenderness on pressure over the ciliary region, and dimness of vision so profound that the patient could only distinguish light from darkness. The history of the case, taken along with the appearances presented by the eye, made it almost certain that the globe contained a foreign body, and this diagnosis was confirmed by the Röntgen rays. The photograph clearly showed the shadow of a piece of metal in the ciliary region. The eyeball was enucleated, and when the globe was bisected, a minute piece of the casing of the cartridge was found in the position indicated by the skiagram.

3. In the above case the application of the Röntgen rays was really unnecessary, for all the circumstances of the accident made it practically certain that the injured eye contained a foreign body. In many instances, however, the X-rays afford very valuable assistance, for they render it easy to demonstrate the presence of a foreign body, whose existence would otherwise be doubtful. The following is a case in point: F. C., 21 years of age, was sent by Dr. Trimble of Strabane to

consult me on the 7th of November, 1900. The patient was a stonecutter, and about the middle of September last, while he, along with other workmen, was hewing a block of granite, a splinter flew from a companion's chisel and struck him on the left eye. He was at once removed to a hospital in Londonderry, where he remained for six weeks, and shortly after he left that institution he was sent to Glasgow. When I first saw him the iris was discoloured, the pupil irregular, the lens cataractous, tension normal, and light perception good. An X-ray photograph revealed a foreign body in the vitreous chamber (Plate 1, fig. 2), and although, from the distinctness of the shadow on the sensitive plate, this was diagnosed to be of metal, the patient himself insisted that it must be a chip of granite. A magnet was applied to the eye, when immediately a sensation of pain was felt, the iris and lens bulged forwards, and a minute particle of metal darted through the cataract into the aqueous chamber, from which it was easily extracted. The cataractous lens was afterwards removed by irrigation with sterile saline solution, and the adhesions between the iris and the capsule were divided by Carter's scissors. The operation was followed by no inflammatory reaction, and a month afterwards the patient was able to count fingers at three feet, and after the remains of the lens capsule occupying the pupil were divided he could easily distinguish large objects, and guide himself about with the right eye closed. When he left for home ten days after the operation his sight was steadily improving.

The magnet employed in this case was one constructed for me by Mr. John Trotter of 28 Gordon Street, Glasgow, and it combines the advantages of the ordinary Hirschberg's electromagnet with the high traction power of Haab's giant magnet. This large portable magnet can, by the attraction of its core, lift a mass of iron weighing 2 cwts.; but the aim in its construction was to produce an instrument which would attract a small fragment of metal a considerable distance, rather than one capable of lifting a very heavy weight. When ready for use it is suspended from the roof of the operating room by two pulleys, and carefully balanced by a counterpoise. Into one end of the core a number of tips varying in size and shape

can be screwed, and when it is arranged in the manner just described, and connected with an electric current controlled by a special switch, its manipulation is quite easy. In practice it is better, however, to have in addition a Hirschberg's magnet at hand, because, if the rough edge of a chip of metal become entangled in the iris, there is great danger that the large magnet will extract it so rapidly and forcibly that a considerable portion of the iris will also be dragged out at the same time. It is better, indeed, after the foreign



body has been displaced from the deeper parts of the eye into the aqueous chamber, to complete the operation for its removal with a less powerful instrument.

4. Important as is the aid given by the Röntgen rays in demonstrating the presence of a foreign body in the eyeball, still more important is the assistance they afford in determining its exact position. The principles underlying the method by which this is accomplished were first described by Mackenzie Davidson, but, as is shown in the figure, my apparatus is somewhat simpler than his both in construction and in manipulation. The patient sits on a comfortable chair

with the head supported by a rest, to the upright of which is attached a curved arm carrying a pocket to receive the sensitive plate. By this means the latter can be brought closely in contact with the parts surrounding the eye to be photographed. The following is a good illustration of the advantages of the method: J. J., aged 36, an ironworker,



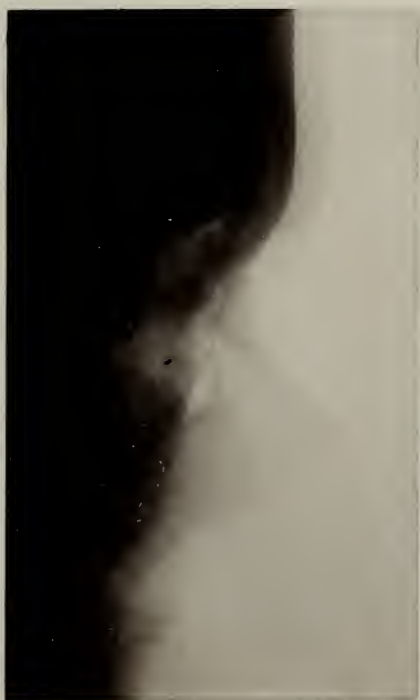
came to the Ophthalmic Institution on 23rd September, 1900. About six months previously he had been struck on the left eye by a chip of steel, but it was not thought that any foreign body had lodged in the globe. There was a well-marked cicatrix at the lower aspect of the cornea, to which the iris was adherent, but the intra-ocular tension was normal, and the light perception good, though the pupil was small and irregular, and the lens cataractous. Although the eye showed no sign of active inflammation, the patient complained that

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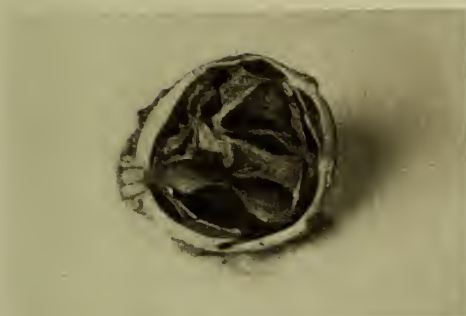
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it always felt irritable, and he had recently begun to fear that this irritation might injure the sound eye. As a small brownish spot, resembling rust, was noticed in the opaque lens, a skiagram was taken, with the result that a fairly large foreign body was detected, and localised in the lower aspect of the eyeball immediately behind the cataract. The patient was put under the influence of chloroform, an incision was made through the cornea, and after an iridectomy had been performed in the position indicated by the skiagram (Plate 1, fig. 3), the chip of metal was easily withdrawn from the eye by the electro-magnet. After this operation the patient recovered rapidly, but owing to the cataract the vision was not much improved. On the 26th of January of this year a capsulotomy was performed, with the result that the patient was afterwards able to distinguish large objects, and to guide himself about with the sound eye shut.

5. The above is an example of a successful operation for the extraction of a piece of steel which had been lodged within an eyeball for nearly six months. Such a fortunate result is, however, not by any means the rule, as in many cases the eye is damaged irreparably from the moment it is struck. The shock, the large size of the foreign body, and the great liability of the vitreous to become infected by septic organisms, all contribute to bring about a disastrous result. Obviously the sooner a foreign body is extracted from the eye after an accident, the greater the chance of preserving useful vision; but in many cases in which the operation has been promptly and successfully performed, the globe is ultimately completely destroyed by plastic cyclitis. The following is an example: On the 18th January, 1901, W. P., aged 21, was struck on the right eye by a chip of steel which flew from the head of a fellow-workman's hammer. He was brought to the Ophthalmic Institution within five hours after the accident, and a piece of metal was extracted by the electro-magnet, without much difficulty, through the original wound in the cornea. After the operation the patient suffered very little pain, and the inflammatory reaction was slight, but light perception steadily failed and in three or four days was lost altogether. By the end of a fortnight

the left eye was sensitive to light and watered considerably, and a few days later the injured eye became tender to pressure and began to soften and shrink. It was enucleated at once, and when the globe, after hardening in formaline, was bisected, it exhibited all the pathological changes associated with well-marked irido-cyclitis. In a short time there would almost certainly have been sympathetic inflammation of the fellow eye (Plate 2, fig. 2).

6. The presence of a foreign body may be demonstrated, and its position within the eyeball accurately localised, by the Röntgen rays, and yet there may be no possibility of extracting it. Such an unfortunate result may occur even although the foreign body be one which the electro-magnet will attract, for it is not at all unusual for a particle of steel to become so embedded in the tissues as to be completely insulated—the more minute the particle the greater this difficulty. The following is a good case in point: On the 16th October, 1900, A. F., aged 22, was sent by Dr. Alexander Stewart to consult me. The right eyeball turned outwards, and its lens was cataractous; and the patient's object in coming to me was to get the eye restored to its proper position and the cataract removed. Ten years previously his eye had been injured by a chip of metal, but although he had been under skilled treatment at the time of the accident the presence of a foreign body within the eyeball had not been suspected. A Röntgen-ray photograph, however, clearly revealed a small piece of metal in the ciliary region, but all attempts to extract it by the magnet failed. The cataract was easily removed, and as soon as the patient had recovered from the operation another skiagram was taken, and the foreign body was found to be in exactly the same position as before (Plate 1, fig. 4). Some weeks later a capsulotomy was performed, and since then vision has gone on steadily improving.

7. In A. F.'s case, although the sight is improved and the deformity lessened, the eye still retains the foreign body, and all experience goes to prove that under such circumstances destructive inflammation may arise at any moment. For years the foreign body may do no harm, and then suddenly and inexplicably there may be such a severe onset of cyclitis that

the eye will be lost, and only by prompt enucleation will its fellow be saved from destruction through sympathetic ophthalmitis. This was the state of matters mentioned incidentally in connection with the first patient referred to in this paper, but the case about to be narrated is probably even more interesting and instructive: On March 20th, 1900, J. H., aged 31, came to the Ophthalmic Institution complaining of recurrent inflammation of his left eye, the sight of which was rapidly deteriorating. He said that when he was about ten years of age he was standing one day in a blacksmith's shop, when he was struck on this eye by a piece of metal. He was skilfully treated at the time of the accident, and apparently made a perfect recovery. At all events, for more than twenty years the eye had never caused him a moment's discomfort, and, so far as he knew, sight was in no way impaired. Quite recently, however, more especially during the night, he had begun to suffer pain in the left eye, and to notice that the sight was failing rapidly. He could assign no cause for the occurrence of these inflammatory attacks, which had become steadily worse as time went on. When he was admitted to the hospital the eye was very painful and tender to touch, there was much circumcorneal injection, the iris was discoloured and tremulous, the pupil small and irregular, the vision very defective, and owing to opacities in the media no details of the fundus oculi could, on ophthalmoscopic examination, be distinguished. For a few days after admission the symptoms abated considerably, but on the night of the 30th March there was a sudden outburst of excruciating pain, accompanied by severe retching and vomiting, and in the morning the aqueous chamber was nearly a quarter full of pus. After this the patient's sufferings were much less, but the respite was of brief duration, and from this time onward he was tormented by constantly recurring attacks of pain and vomiting followed by the appearance of pus in the anterior chamber. In the intervals there was very little discomfort, but the eye was quite blind, and on the occurrence of a very severe relapse during the month of August the patient readily consented to enucleation. After the globe was hardened in formaline it was bisected, when it was seen that the lens was

dislocated, and the ciliary body and iris were inflamed, and as a probable cause of all the trouble a small fragment of steel was found firmly embedded in the ciliary region (Plate 2, fig. 3).

In this series of cases failures have been recorded as well as successes, and the latter are unfortunately few. Before, however, the magnet and the Röntgen rays were introduced into practice, there is not, as far as I am aware, a single instance on record of a foreign body having been removed from the vitreous chamber, and the sight at the same time saved. We may, therefore, justly regard the preservation of useful vision after such accidents as those I have referred to as one of the greatest triumphs of modern Ophthalmic Surgery.

TWO CASES OF NERVOUS DISEASE: (1) A CASE OF PERIPHERAL NEURITIS WITH SYMPTOMS OF BULBAR PARALYSIS. (2) A CASE OF LOCOMOTOR ATAXY ASSOCIATED WITH PROGRESSIVE MUSCULAR ATROPHY.

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(1) A CASE OF PERIPHERAL NEURITIS WITH SYMPTOMS OF BULBAR PARALYSIS.

G. M., aet. 29, a farmer, was admitted to Ward 7 of the Royal Infirmary, on January 14th, and he died on January 21st, 1896.

The commencement of the illness seems to have dated back to January, 1895, when the patient had a sore throat lasting for several days. It was not at the time thought to be diphtheritic, and there was no diphtheria known to be in the neighbourhood. But some weeks later the patient found that he had difficulty in masticating his food, and he was quite sure that this was due to loss of power in the muscles of the jaw. In the month of March the soft palate became involved, giving rise to a nasal quality in the speech and allowing fluids to regurgitate through the nose. The eyelids were likewise affected, for they could neither be firmly closed nor completely raised, and the patient had the appearance of a slight double ptosis. These symptoms were more marked in the right than in the left eye. At this time he also complained of his legs and arms being "weak." He said he was easily tired, that his legs would give way under him and that he several times fell.

This condition of the limbs, however, improved with electrical treatment and he was ultimately able to walk quite well.

For some months prior to admission there was difficulty in swallowing, which seemed to gradually get worse. More recently loss of power in the lips and tongue developed, and there was difficulty in chewing and controlling the food while in the mouth. At the time of admission to the ward the tongue, though it presented no apparent atrophy, could not be protruded to any extent and its movements within the mouth were very limited. Neither could the lips be closed even by a strong effort, and saliva was always dribbling from out the corners of the mouth. The patient could make no whistling sound and when he attempted to do so the air escaped freely from between the lips. The lower jaw seemed to be too loosely articulated, it tended to fall forwards, and the patient seemed to find it impossible to keep the upper and lower dental arches in apposition. At this time he could still swallow, though with difficulty, and his speech, while indistinct, was quite intelligible. But during the week subsequent to admission the symptoms rapidly got worse, so that by the 18th inst., he had to be fed by the stomach tube and his articulation was so much affected that he could only be understood when he conveyed his wishes in writing. The respiratory movements by this time were mostly diaphragmatic, although on admission to the ward there was still considerable intercostal action. Finally the breathing became entirely abdominal and death seemed to supervene from failure of respiration.

While in hospital there was little loss of power to be noted in the muscles of the arms and legs. The grasp in both hands was good, and the patient could stand with his feet together and the eyes shut without any difficulty. He could walk quite well, and while the gait was slow and deliberate, it presented no characteristic abnormality. The tendon reflexes were normal and there was no ankle clonus. The plantar and cremasteric reflexes were not obtained, but the abdominal reflex was active. There was no reaction of degeneration to be found in the muscles of the face, arms, shoulders or legs. There was no impairment of general sensibility and the special senses seemed quite normal. The movements both of the internal

and external muscles of the eyeball remained normal. The patient complained of "weakness" in his back and he seemed to have difficulty in raising himself from the dorsal decubitus without first turning on to his side. One could not, however, be certain that this symptom was due to actual paralysis in any of the muscles and the trunk.

The heart and lungs showed no signs of disease, but the urine always contained a certain amount of albumen.

At the *post-mortem* examination the macroscopic appearances were found to be perfectly normal, except that both kidneys were seen to be in the early stage of interstitial nephritis. The cord, medulla, pons, and the ixth, xth, and xiiith nerves were examined microscopically by Weigert's and Nissl's methods. The right and left vagus and right and left glosso-pharyngeal nerves showed a fairly well-marked degeneration, with great increase of the internodal nuclei. Both right and left hypoglossal, however, seemed quite normal. Weigert's staining showed no degeneration in the motor tracts of the cord, medulla, or pons; and the ganglion cells in the anterior horn of the cord, in the hypoglossal nucleus, the nucleus ambiguus, and in the facial nucleus seemed little altered. These cells throughout were well shaped, they had central nucleus, and there was no apparent absence of processes. The Nissl bodies, however, were not so well defined as might be desired, but there was certainly no advanced chromatolysis to be seen; and if there was any fragmentation of the Nissl granules, it was very slight. The defect in the definition of these Nissl granules was probably due to some slight error in fixing or in the stain itself, but examination of a large number of sections of the cord and medulla convinces me that the ganglion cells of the anterior horns of the xiiith nucleus were practically normal. One cannot speak with the same assurance of the cells of facial nucleus and the nucleus ambiguus. There were not the same number of sections at these levels to examine, and it is difficult from a few transverse sections (especially in the case of the xth nucleus) to give a definite opinion as to the condition of the whole nucleus. We may take it, however, that the cells of the viiith and xth nuclei showed little, if any, change.

Microscopic examination of the muscles of the pharynx and tongue showed them to be quite normal.

Viewing this case, then, in the light of the *post-mortem* examination, it seems, with little doubt, to be one of peripheral neuritis, probably of diphtheritic origin. The microscopic appearances explain the clinical signs and symptoms, except that it is difficult to understand the absence of degeneration in both hypoglossal nerves. The presence of at least a certain amount of paresis in the tongue seemed undoubted during life; but, on the other hand, it is to be noted that careful microscopic examination of that muscle showed no corresponding degeneration in its fibres, such as one would look for in a case of peripheral neuritis. Possibly, as is so often the case in diphtheritic paralysis, only some of the fibres in the nerve were affected, and these towards their distal terminations, where degeneration most often shows itself. It is, therefore, much to be regretted that both hypoglossals were not examined through a greater extent of their distribution, and stained also by Marchi's method. It is quite certain, however, that in the portions of the nerves examined there was no increase of the internodal nuclei, and that the axis cylinders were quite intact. These points were well shown in the sections stained with methylene blue and Congo red.

If this case, then, is one of peripheral neuritis, its special interest would seem to lie in the fact that, with histological changes confined to the peripheral nerves, we have a grouping of symptoms almost identical with those found in bulbar paralysis. As in that disease, the onset was gradual. The lips, tongue, soft palate, pharynx, and larynx were all involved. It is true that the lips and tongue were affected late in the disease, and that there was no atrophy of the tongue. But it sometimes also happens this way in bulbar paralysis. In bulbar paralysis, too, we sometimes have the muscles of the upper part of the face and of the jaw affected just as in this case. The symptoms, therefore, closely resemble those found in bulbar paralysis; and the above record may be of value in that it might suggest an explanation of these rather obscure cases of "chronic bulbar paralysis without anatomical lesion."

(2) A CASE OF LOCOMOTOR ATAXY ASSOCIATED WITH
PROGRESSIVE MUSCULAR ATROPHY.

J. O'B., aet. 48, a labourer, was admitted to Ward 7 of the Royal Infirmary, on June 19th, 1896.

The notes of this case are rather scanty as the patient died within twenty-four hours of admission. He had been admitted a month before to a surgical ward on account of a bruise on the right hip, and he was transferred to the medical house as a case of progressive muscular atrophy. During his residence in the surgical wards his symptoms seem at no time to have suggested a diagnosis of locomotor ataxy.

The history the patient gave was that for ten years past he had been aware of atrophy in his hands, which had gradually been getting worse. On examination both hands presented the typical "bird-claw" appearance. The thenar and hypothenar eminences were quite gone and there was much atrophy in the interossei muscles. The upper-arm muscles were also involved to a certain extent. There was considerable loss of power in both hands and arms, and tremor in the hands was present. The tactile sense was much impaired in the parts of the hands supplied by the ulnar nerve.

The muscular power of the legs was good, but the tactile sense was lost in both feet and much diminished in the legs as high up as the knees. The plantar and knee reflexes were gone, but the cremasteric and abdominal ones quite active.

The pupils were unequal and neither contracted to light. The sense of smell was somewhat deficient, but the senses of sight, taste, and hearing were quite normal.

For a period of four years patient voided his urine in bed, but he said he could retain it during the day. He had been a heavy drinker for many years, and there was trembling of the lips and tongue, especially when he was speaking. The memory, also, was somewhat impaired.

On May 23rd delirium tremens developed and lasted for five days, in spite of treatment by large doses of chloral and bromide of potassium. This illness left him very feeble.

Shortly after admission to the medical wards convulsive

seizures supervened. These were clonic in character, and generalised, but they affected the extensor more than the flexor muscles of the body. The duration of each convulsion varied much, the longest being three hours and the shortest about one minute. These seizures continued, on and off, for about twenty-four hours, at the end of which time the patient died.

The *post-mortem* examination revealed infarctions in the left kidney and a condition of interstitial nephritis in the right. There was some excess of fluid in the soft membranes of the brain, and some ecchymosis on the right frontal lobe and over the left motor area on both sides of the fissure of Rolando. Otherwise the macroscopic appearances were practically negative.

Microscopic examination.—The nerves of both arms were stained with osmic acid and with carmine. They all showed marked degenerative changes, with great increase of the inter-nodal nuclei and connective tissue elements, some of the nerves being just like strands of fibrous tissue.

The cord, medulla and pons were stained according to the methods of Weigert and Nissl. With the *Weigert* stain there was seen to be a well-marked degeneration in the posterior columns of the cord. In the lumbar region this involved both the postero-external and the postero-internal columns; but passing upwards it gradually confined itself to the postero-internal till in the cervical region it was only this tract that was affected. The posterior roots in the lower cord were much sclerosed but in the cervical region they were apparently unaffected. The anterior roots in the cervical region seemed practically normal. There was no apparent degeneration in any of the antero-lateral tracts, neither did the motor fibres in the medulla or pons show any abnormality.

In the specimens stained by Nissl's method special attention was paid to the appearances of the motor ganglion cells. Sections were examined from six different levels of the cervical cord, four of these being from the cervical enlargement, and the most striking feature of these sections was the small number of cells showing any marked pathological change, the vast majority being well shaped, with central nucleus, and their

processes intact. Many of them contained a considerable amount of yellow pigment, but in only a very small proportion was anything like a chromatolytic change to be seen. A count of the cells in the anterior horns was made, and although the number in all the sections through the cervical enlargement was below that of the normal cord, still the number of cells present was much greater than one would look for with such an extreme degree of muscular atrophy as was present in this case. The mesial group of cells was not considered in the count, and the antero-lateral and postero-lateral groups were as a matter of convenience taken together as one. The average number of cells in this lateral group throughout the cervical enlargement was 36.6 as against 54, the average number found in a healthy young adult. But it is to be here noted that there was also a diminution in the number of cells in the anterior horns in the lumbar region (30 as compared with the normal 47), though there was no apparent corresponding atrophy of the muscle-fibres supplied by these cells. I take it, then, that in estimating the number of ganglion cells in such a case as this—a man, aged 48, and who, from our point of view, was really much older—we must not compare his cord with that of a young adult, but rather with that of a much older man and one who has not recently been living an active life; and I find in counting the cells in other cases that the standard of the young adult is too high for most of the cases over 50 years that one examines.

I take it, then, that in the cervical enlargement there was no atrophy of ganglion cells corresponding to the very great atrophy in the muscles of the hands and arms, or to the degree of degeneration found in the nerves supplying these muscles. The absence, too, of degeneration in the antero-lateral tracts of the cord, and in the corresponding tracts of the medulla and pons corroborates this view. And so the conclusion that is forced upon us is that the primary lesion has been one of peripheral nerves. But this corresponds with the researches of Dégérine which point to the muscular atrophies which sometimes complicate locomotor ataxy, as being usually due to degeneration in peripheral nerves, though it is opposed to

the view of Gowers, who tells us that any extensive muscular atrophy found in that disease "is probably due, as a rule, to degeneration in the anterior cornua."

From this point of view, then, this case of ours has presumably been primarily one of locomotor ataxy, and the progressive muscular atrophy a complication. But on the other hand, it would be impossible to insist that it was not the progressive muscular atrophy that was the primary disease, for the atrophy was always the most outstanding feature of the case, and it had been present for at least ten years. It is quite conceivable, however, that locomotor ataxy may have been present for a longer period still, and yet not have obtruded itself specially on the notice of a patient whose habits of life would not lead to accuracy of observation, and whose mind for so many years was fixed on the atrophy and loss of power in his arms and hands.

A CASE OF POST-RECTAL DERMOID, REMOVED BY
THE PARASACRAL ROUTE: WITH REMARKS
ON TUMOURS OF THE SACRO-COCCYGEAL
REGION.

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MRS. B., aged 34, was admitted to Ward 26 of the Glasgow Royal Infirmary on the 27th December, 1899, with what was supposed to be, and what I myself regarded as a fistula in ano of many years' standing. She stated that at the birth of her first and only child, 10 years before, a few days after leaving bed she began to be aware of a hard painful swelling in the right buttock; this was incised by the medical man in attendance and gave escape to a large quantity of dark-coloured material. The opening had continued to discharge almost continuously ever since. Later on, she told us further, that as an infant she had had a swelling in the same situation which also was incised, but which closed after a time.

On examination, there was found between the anus and the right tuber ischii, a warty-looking granulating surface about the size and shape of a haricot bean; from an opening near its middle there escaped on pressure a small quantity of clear glairy fluid. There was some induration about the raw surface, and slight tenderness was noted. A probe passed into the opening travelled upwards and inwards, and met with resistance after about two inches of probe had been swallowed. With a finger in the rectum, the point of the probe could be felt quite outside the mucous membrane of the bowel about three inches from the anal orifice, the impression conveyed

being that some force would be required to bring it into the lumen of the bowel.

On the 8th January, the patient having been put under chloroform, I laid open the sinus in the direction in which the probe travelled, which was backwards and upwards. I then thought I had come to the end of the cavity, one, namely, in the ischio-rectal fossa; and suspecting the nodular mass in the skin to be tubercular, I proceeded to excise it and the walls of the sinus, noting that the probe had not passed within the pelvic fascia. On cutting away the indurated tract behind and above, it was seen that the section was densely fibrous, and from the upper end of the wound there leaked a thin ill-smelling discharge. An incision into the tissue beyond gave escape to this fluid in large quantity, and the opening was found to communicate with a smooth-walled cavity behind and to the right of the rectum, into which the index-finger went its full length between rectum and sacrum. This opening was in the levator ani and the layers of the pelvic fascia; it was enlarged so as to permit of the cavity being wiped out and stuffed.

So far I had no idea of what the nature of the case really was. I was inclined to regard the cavity as due to chronic abscess, possibly connected primarily with the rectum, or possibly due to a pelvic cellulitis starting from the neighbourhood of the cervix uteri. I even thought of a salpingitis. About six weeks later, when the wound had contracted without diminution of the discharge, I re-opened it and scraped the cavity freely. This was followed by as little improvement; and after this I became convinced that I had to do with a definite cyst of some kind, and the idea of a dermoid came to my mind. I had not made any examination of the scrapings, but perhaps the bitter offensive smell had something to do with suggesting the presence of epithelial débris. On 14th March, 1900, with the kind assistance of Mr. H. E. Clark, I operated by Kraske's method, removing the coccyx and right lower angle of the sacrum. After getting through the levator ani, the wall of the cyst was exposed and somewhat tediously dissected out. The dissection was quite delicate on the side next the rectum, and at the last I applied a ligature before cutting it away. The

cyst itself was about the size of a Tangerine orange, and the cavity left, a rather unpromising one to treat. It could not be filled with soft tissues and had to be stuffed, the skin wound being closed only in part.

The patient was kept lying on her face and the utmost care taken to prevent soiling of the dressings, but by the second day there was a rise of temperature and obvious inflammatory symptoms. Frequent dressings had to be resorted to, and after some time, the cavity having become clean, the wound was further closed by suture. Finally, the remaining raw surface was grafted, but it was not till the 19th June that the patient was dismissed.

She has been seen since (April, 1901), with a healed depression to the right of the lower end of the sacrum, but with no material damage to the floor of the pelvis, though on coughing or straining there is a somewhat diffuse bulging of the scar.

Microscopic examination of the cyst wall, which was kindly undertaken by Dr. Workman, Pathologist to the Infirmary, showed a fibrous-tissue structure with striated muscle fibre, no doubt representing portions of the levator ani, lined by a stratified more or less squamous-celled epidermis. In the fibrous tissue were embedded glands of a convoluted tubular structure resembling sweat glands.

Post-rectal dermoids are to be distinguished from rectal dermoids, and both from dermoids of the peritoneal cavity (ovarian for the most part) bursting into the rectum. Secondly, they are to be distinguished from tumours of a more complex structure, peculiar to the same region, the compound cystic sarcomata of Braune (*Die Doppelbildungen und Angeborene Geschwülste der Kreuzbeingegend*, 1861) or thyroid-dermoids of Bland Sutton, whose origin in the post-anal gut or neurenteric canal of the embryo was first propounded by Middeldorpf (Virchow's *Archiv*, Bd. 101, S. 37).

Mr. Bland Sutton refers to three cases of rectal dermoids, and says "a feature of these cases is that the tumours are furnished with long locks of hair, which protrude from the anus and annoy the patient. Like post-rectal dermoids, they sometimes contain teeth." (Port, *Trans. Path. Soc.*, London,

xxi., p. 307; Clutton, *Trans. Path. Soc.*, xxxvii., p. 252; Danzel, *Langenbeck's Archiv*, Bd. xvii., S. 442.) In the last-mentioned case the tumour, which was carefully described, was evidently complex in structure. It was removed from a woman 25 years of age, and is said to have contained brain-substance and bone, besides bearing on its surface hair and a tooth. The patient died three months after the operation, of what is described as a local peritonitis. The operation was done after dilatation of the anus, and was evidently a severe one, contrasting with what is required when such a tumour is pedunculated. The case reminds us that rectal dermoids are not always pedunculated.

"The removal of pedunculated open dermoids of the rectum," says Senn (*Tumours*, 2nd ed., p. 650) "offers no difficulties; on the contrary, the extirpation of perirectal tumours requires often a formidable operation. Usually the difficulties of operative removal are increased by inflammation and suppuration, which render the dissection tedious and difficult." He refers to "a case of post-rectal dermoid which had suppurated and ruptured just below the coccyx. . . . There was found an opening large enough to admit three fingers, lined by skin and leading to a cavity the size of a child's head lined by hairy skin. Decision was against interference . . . the writer could hardly imagine how such a cavity could be made to heal after dissecting out the entire sac." Such a case is narrated in detail by Deahna (*Archiv für Gynaekol.*, Bd. vii., S. 305). The patient was a girl of 18, who suddenly developed a tumour in the lower abdomen with pressure on the rectum and retention of urine. The lower wall of the cyst was felt between the rectum and sacrum, the upper impinged on the abdominal wall between the umbilicus and symphysis. Exploratory puncture per rectum was followed by suppuration and acute distension, resulting in sloughing of the soft parts including vaginal wall and urethra, analogous, as is pointed out by the writer, to what takes place in protracted labour, from pressure of the foetal head. Here also the patient, who was under the care of Czerny, had to be dismissed relieved of the pressure symptoms but with an open cyst cavity.

Trzebitzky (*Wiener Medicin. Wochenschrift*, 1885, p. 393)

reports a case from Mickulicz's clinic (Cracow) where two separate but connected cysts were present—the one presenting externally in the left buttock, of the size of a child's head; the other, the size of an orange, occupying the left half of the pelvis. They contained a thick yellow fluid with epithelial débris and hairs. The upper one was found to have separated the fibres of the levator ani and to be intimately connected with that muscle. The tumours were dissected out and left a cavity in the pelvis more than the size of two fists. In spite of every care and the use of buried sutures the cavity suppurated and healing was slow. Trzebitzky suggests temporary packing of the rectum in order to secure apposition of the anterior wall of such a cavity to the sacrum.

The same writer is inclined to refer such tumours to errors of development, foetal inclusion in the sense of Cohnheim's theory, in connection with the invagination of the surface epithelium which gives rise to the proctodeum, that is to say, the invagination by which the anus is formed.

The theory which invokes aberrant processes in connection with the proctodeum, while plausible in reference to such cases as the one I have described, is restricted in its application, a restriction which is the more marked as we consider the variety in which congenital tumours develop in the region of the sacrum. Errors of the proctodeum may be invoked to explain simple dermoids—tumours lined by stratified squamous epithelium with or without hair or even sweat glands—but can say nothing to the presence of teeth, still less of bone; nor do they in any way explain tumours whose elements conform to the hypoblastic type. Further, it is to be pointed out that the connection of sacral tumours, cystic or otherwise, is in a proportion of cases rather with the spinal cord than with the rectum, and in these the proctodeum theory is still more obviously at fault. In this connection it is worth noting from a clinical point of view that, as Senn points out, "differentiation of sacral dermoids and of spina bifida is often very difficult, and conclusions should be postponed in doubtful cases until an exploratory puncture has demonstrated the character of the contents of the sac."

So Mr. Bland Sutton (*Tumours*, p. 470), "All reported cases of the successful removal of the sac of a spina bifida from

adults should be carefully studied, because in some instances they may have been dermoids." This is the converse of Senn's statement, and I am able to amplify it by the case of a lymph cyst or hygroma removed by me in the Royal Infirmary from the lumbo-sacral region in a boy of ten. The case had been refused operation elsewhere as a spina bifida, but the tumour, which consisted of three principal sacs connected centrally, was quite superficial to the lumbar aponeurosis.

Among the structures leading to the inclusion of surface epidermis and the formation of true dermoids, there falls to be considered the post-anal dimple or foveola retro-analis. For an account of this structure and its relation to the termination of the spinal cord, I would refer to a paper by MM. Tourneux and Hermann in the *Journal de l'Anatomie and de la Physiol.*, vol. xxiii., "Sur la persistance de vestiges médullaires coccygiens pendant toute la période foetal chez l'homme, et sur le rôle de ces vestiges sur la production des tumeurs sacro-coccygiennes congénitales." Their researches have led these writers to the conclusion that the formation of the post-anal dimple is due to the comparative ascent of the lower termination of the spinal cord caused by the disproportionate growth in length of the spinal cord and the spinal column, and consequent dragging in of the soft tissues with which in its early embryonic stages the cord is in immediate relation. More than one such puckering of the surface may be present, and there may result merely funnel-shaped depressions, or cysts more or less completely shut off from the surface.

Professor Cleland (*Journal of Anat. and Phys.*, Vol. xvii., p. 290) calls attention to the co-existence in cases of well marked dimple of a straight and unduly short sacrum, and is of opinion that both the dimple and the deformity of the sacrum are to be related to an arrested development of the notochord.

The origin of sacral tumours is further complicated by the cases in which we have to do with an unmistakable adherent foetus, and which shade off into those where we seem to recognise more or less well-defined parts, organs or tissues, belonging to a second foetus; and this complexity is increased

by the occasional occurrence of malignancy in some of these tumours, taking the form of sarcomatous or carcinomatous growth with metastases.

Hennig, in Ziegler's *Beiträge*, Bd. xxviii., Hft. 3, gives a valuable review of the theories which have been propounded as to the origin of congenital tumours of this region. Middeldorpf, Ritschl, and Nasse he cites as the exponents of the post-anal gut theory, to whom we must add in this country Mr. Bland Sutton (*Tumours and Dermoids*). Hennig himself is of the opinion set forth by Förster, Virchow, and Ahlfeldt, which ascribes to a foetal implantation almost all tumours of the sacro-coccygeal region, which view has also been propounded by Braune (*Die Doppelbildungen und Angeborene Geschwülste der Kreuzbeingegend*, 1861), and in a Paris thesis by Calbet (1893). The summing up of the last-mentioned author is, that the majority of congenital tumours of the sacro-coccygeal region, which are known in literature under the most varied names, belong to one and the same group, and are to be regarded as dwindled (*verkümmerte*) parasitic embryonic deposits (*Anlagen*).

These tumours, which have all a more or less uniform character, possess mostly a very complicated structure. They contain the most-various tissues, and may present complete organs or fragments of organs which take their origin in a new foetus to a certain extent grafted on the first, or, as it may be otherwise put, incompletely budded from the first.

The other group of congenital neoplasms of the sacro-coccygeal region, according to Calbet and Hennig, comprises those formations which are to be referred to a disturbance of development, development of a particular structure being in the direction of (1) excess or (2) defect (restriction). To the former class belong the cases of tails, with actual bony vertebrae or of mere tail-like structures, such as have more often been described; to the latter (those due to restricted development) belong all cases of spina bifida, different kinds of dermoid cysts due to the driving-in and cutting-off of processes of the external skin, fistulae, etc.

Reverting once more to the subject of the post-anal gut and its relation to this group of thyroid-dermoids or compound

cystic sarcomata, I desire, while recognising the importance of the observations which concern that structure, to dissent from the suggestion put forward by Mr. Bland Sutton, as to its being represented in the adult by the coccygeal body, or Luschka's gland, and the consequent inclusion of Luschka's gland as an originator of this group of sacral tumours. Hennig (*l.c.*) quotes Virchow as having, as far back as 1861, recognised that Luschka's gland had been found co-existing independently in the presence of the tumours in question. Further, while disclaiming any original anatomical observations on the subject, I think it is open to doubt whether what is known of Luschka's gland (cf. the account of it given in Quain's *Anatomy*) entitles it to be regarded as a structure of epithelial character—that is, of epiblastic or hypoblastic origin.

CARDIAC ARRHYTHMIA IN RELATION TO CEREBRAL ANAEMIA AND EPILEPTIFORM CRISES.

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THE occurrence of epileptiform seizures in association with cardiac irregularity is by no means infrequent, if we may judge from the literature of the past twenty years, but it is probably comparatively rare in the experience of the individual clinician. And, in view of this fact, it has been thought that the record of a case which presented some remarkable features might be not without interest. The selection of a suitable title has been somewhat difficult, as the majority of distinctions under which the subject has been treated by various observers have given rise to not a little confusion, and, in a few cases, to entire misapprehension. Among the many designations which have found a place in the recent medical journals, we find such as the following:—"Bradycardia and Epilepsy," "Unusually Slow Pulse and Epilepsy," "Epilepsy of Cardio-Vascular Origin." For reasons which will emerge in the course of this paper neither of the two former titles appears to be sufficiently explicit, and probably both are inaccurate. Of the other it may be said that conciseness has been secured at the expense of clearness. The records which form the basis of this paper have been entirely drawn from one case, but that the observations were neither of an isolated nor casual nature may be gathered from the fact that the patient was under observation for a period of nearly two years, and during that

time offered abundant opportunity for the confirmation or correction of previous observations.

The original intention with which the article was undertaken was to endeavour to make a clinical application of Kussmaul and Tenner's experimental work on the causation of epileptiform convulsions, by inducing cerebral anaemia either mechanically or surgically, and that will be the chief object of the paper; but during the progress of the case several other points emerged which call for consideration. There appears to be almost a unanimity among authors who write upon the subject, that when cardiac derangement is associated with convulsions the former stands to the latter as cause to effect; but comparatively few of the contributors to the literature of the subject have accompanied their reports by sphygmographic evidence, which conveys a comprehensive idea of the chief points which they endeavour to demonstrate, and in view of this the accompanying sphygmograms may not be without value. There are two other points upon which anything but unanimity prevails. In the first place the question arises, does true bradycardia or slow pulse exist in association with convulsions? Secondly, when a slow rate of pulse obtains and is associated with convulsions is the action of the heart altogether in abeyance between the powerful contractions, which have a radial expression, or is it making regular although very feeble efforts to contract? Kussmaul and Tenner's work may not possess the attraction which attaches to novelty, but it has the merit of remaining, although not altogether unchallenged, still quite uncontroverted; and in order to a clearer understanding of the subject, some of the more important conclusions arrived at by these observers will be briefly recapitulated.

"The convulsions appearing in profuse haemorrhage (of warm-blooded animals) resemble those observed in epilepsy."

"When the brain is suddenly deprived of its red blood, convulsions ensue, of the same description as those occurring subsequent to the ligature of the great arteries of the neck."

"Epileptic convulsions are likewise brought on when the arterial blood rapidly assumes a venous character, as, for example, when a ligature is applied to the trachea."

"It is highly probable that in these cases the attack of spasms depends upon the suddenly interrupted nutrition of the brain. It is not caused by altered pressure which the brain undergoes."

"The brain of warm-blooded animals can only be deprived of red blood for a short time, otherwise it loses its capability of resuming its functions when again supplied with the nutritive fluid, and the appearance of death becomes a reality. The brain of some rabbits preserved this capability for two minutes."

"Suddenly withheld nutrition is only one of the causes by which the brain is brought into that peculiar internal condition which is manifested in the form of an epileptiform attack."

The writer desires to acknowledge his indebtedness to Professor Samson Gemmell for permission to publish the clinical records of the case.

CASE I. Observed by the Author.

W. T., aet. 48, a weaver by occupation, came under observation in the Western Infirmary, Glasgow, on September 23rd, 1894, with the complaint of taking what he called weak turns, associated with palpitation, sensations of giddiness, and occasional loss of consciousness, which symptoms had existed for some two weeks previously.

Personal History.—He had never been incapacitated for work by illness at any period of his life prior to the onset of his present illness. There was no history of syphilis, and he had never had rheumatism in any form. In 1892 he suffered from what he called indigestion, and experienced palpitation at night, but this symptom did not obtrude itself upon him during ordinary or unusual exertion. *Occupation.*—In his work as a weaver he was not subjected to any great physical strain, either continuously or otherwise.

History of Present Illness.—About two weeks prior to his admission to the hospital, and while he was standing at his loom and stretching over to tie a thread, he suddenly experienced a sensation of faintness, and a feeling as though he would fall. He remembers nothing of what followed after that for

some seconds apparently. But when consciousness returned he felt that his face was flushed, and his heart was beating forcibly. He was removed to his home, where he remained in bed during the two weeks prior to his coming under observation. With regard to the actual symptoms incidental to this first attack no reliable information can be obtained, but his own feelings and the observations of his friends would seem to indicate that it was almost identical with subsequent seizures. During the interval which elapsed between the occurrence of his first attack and his coming under observation, he has had many similar seizures. Sometimes they seemed to have followed one another in rapid succession, at others whole days have elapsed without the occurrence of a single attack. With regard to the description of these seizures, the information of his wife (who was present at the time of the drawing up of the initial report) is not very full; but she saw him during a seizure after admission to hospital, and she said the attack she then witnessed was identical with those which occurred previously.

When the patient was first seen, the striking fact was the extreme facial pallor, and indeed the patient appeared to be dead, and on palpation of the radial artery no pulse was perceptible. Then one or two beats became perceptible, and gradually increased both in frequency and volume; at the same time the extreme pallor of the face and lips was replaced by very marked flushing. The patient opened his eyes and looked about in a dazed manner as if wondering where he was.

From the sphygmographic tracings which were then taken, it will be seen that there were long intervals during which no pulse was appreciable at the wrist. These intervals lasted sometimes for ten or fifteen seconds. If the finger were kept on the pulse, the following facts were observed and noted in order of their appearance. First, the pulse became suspended; then, and almost at the same moment, the face became rapidly and extremely pale. If the period of asphyxia (here the word asphyxia is used as indicating the absence of the pulse at the radial artery as appreciable to the finger) were only of short duration, say of four or five seconds, there was a gradual and crescendo return of the pulse and a coincident flushing of the face. During these short pauses the patient appeared to be

merely sleeping. Frequently, however, the period of asphyxia lasted much longer, ten, fifteen, and twenty seconds elapsing between the pulse beats. During this time, commencing with the disappearance of the pulse, the pallor of the face became more and more marked; but during the earlier portion of the period of asphyxia nothing special was noted, the patient simply presenting the appearance of death, or breathing in a very shallow manner. Later on, however (perhaps after some fifteen seconds of asphyxia), spasmodic twitchings of the muscles of the face were observed; then, as the period lengthened, the spasmodic movements extended to the muscles of the neck, frequently carrying the head to one or other side, and ultimately the arms and legs took part in the convulsive movements. During the more severe crises, either concomitant squint of both eyes or conjugate deviation to either side was observed. Frequently the eyeballs were turned in an upward and inward direction, and the pupils became widely dilated. When the convulsion was at its height, respiration was noisy, with considerable puffing out of the cheeks and lips and the appearance of froth at the mouth. During the greater part of the period of asphyxia the patient was quite unconscious. The return to consciousness was preceded by the noisy breathing and very marked flushing of the face. On the termination of the convulsion, the patient would look about him in a dazed manner as if just aroused from deep sleep. The flushing of the face and the return to consciousness were always almost simultaneous with the appearance of the pulse at the radial artery, the pulse being felt slightly before the flushing of the face was observed. Auscultation over the precordium, during the asphyxial pauses, discovered what appeared to be abortive beats of the heart, but these beats were not appreciable to the finger at the radial pulse, or indeed in any of the superficial arteries.

Nothing of the nature of Cheyne-Stokes breathing was observed during the crises. During the ordinary or powerful cardiac contractions the second sound, as heard over the pulmonic area, was accentuated.

Examination of the Heart. Area of Precordial Dulness.—The upper border was situated on the fourth left rib. The

left border was just within the nipple line. The right border was on the left margin of the sternum. The apex beat (during the powerful systoles) was visible and tangible in the fourth and fifth inter-spaces, with the point of maximum intensity in the fifth space, about half an inch to the inner side of the vertical line of the nipple.

Lungs.—Percussion and auscultation detected nothing in either lung calling for note.

Liver.—Dulness measured four inches in the vertical nipple line.

Urine.—Was dark amber in colour, with no sediment. The specific gravity was 1015, the reaction was acid, neither albumen nor sugar was detected by the usual tests.

September 25th.—Since admission the paroxysms of asphyxia have been of very constant occurrence, and alternating with periods during which the cardiac action was perfectly regular. A number of tracings taken on the two days following admission showed long periods of asphyxia, the pauses in some cases lasting as long as 15 seconds; one tracing taken during the space of one minute indicated three periods of asphyxia, each of from 12 to 15 seconds' duration, and three periods during which pulsation in the radial artery was recorded—altogether 18 beats being registered in 60 seconds (see tracing No. 2). Another series of tracings taken on the same date showed much shorter periods of asphyxia, 7 or 8 beats being registered in half a minute, and 11 in a whole minute. Most of these beats occurred at regular intervals of 5 or 6 seconds. The terminations of the more lengthy periods of asphyxia were frequently, if not usually, associated with convulsions which occurred when the facial pallor was most profound, and ceased coincidently with the appearance of the facial flushing, and of the radial pulse. During the earlier part of these pauses gradual loss of consciousness was noted, and continued until, and during, the convulsion. The convulsions were, for the most part, more severe than formerly, involving not only the face and neck, but extending to the upper and lower limbs.

Yesterday (*September 24th*) the periods of asphyxia being of frequent occurrence, strychnine in doses of $\frac{1}{50}$ of a grain

was administered hypodermically; brandy in ounce doses given by the mouth; and a synapism applied over the precordium. In the course of half an hour after this treatment was instituted the pulse was found to have become perfectly regular both in its volume and rhythm; the tension was also good.

To-day (*September 25th*).—Regularity of the pulse continued all day, and no tendency to convulsive seizure was observed.

September 26th.—Irregularity of the pulse was again noted this morning, when stimulants and strychnine were administered. As the patient appeared to be in a very critical condition, no sphygmographic tracings were taken at this time. In the course of the afternoon there was a marked improvement in the patient's general condition. The pulse became perfectly regular, of good volume, and of excellent tension, as indicated in tracing No. 4.

September 27th.—This morning a new feature presented itself in connection with the character of the pulse. Periods of regularity of volume and of rhythm alternated, with periods when the "pulsus bigeminus" was well demonstrated by means of the sphygmograph (see tracings Nos. 5 and 6), and readily appreciable to the finger. In the afternoon the pulse again became very irregular, the asphyxial pauses were frequent and prolonged, and the patient was practically passing out of one spasm into another. Indeed, the patient appeared to be dying.

September 28th.—The patient's general condition indicates improvement. On auscultation over the precordium, what appears to be a triple rhythm of the cardiac sounds is noted, the first sound apparently being doubled. This fact was observed chiefly over the lower end of the sternum and during the forcible cardiac beats, while during the feeble beats the two reduplicated sounds were audible, but not the single (that is, it was supposed that the doubled first sound was audible, but not the second).

September 29th.—The pulse, as counted at the wrist by the finger, numbered 20 beats to the minute as a rule, but sometimes only 16 beats were registered, and from 16 to 24 were about the limits of variation. Sphygmograms taken to-day also demonstrate characters not previously observed; hitherto no break in the down-stroke or diastolic part of the tracing

was shown, whereas to-day very slight but quite unequivocal waves were registered on the descending line of the tracing. The number of these waves varied according to the length of the so-called diastole or "asphyxial pause," sometimes only two waves appearing, at others as many as six being recorded. That these waves were not the result of either the faulty adjustment of the instrument or tremor of the patient's hand seems evident from the fact that on auscultation over the heart, feeble but distinct cardiac sounds could be heard synchronously with the appearance of the waves on the tracing; and, further, these small waves were too well defined and occurred at such regular intervals as to preclude the possibility of their being due to any extraneous influence. On counting the powerful and feeble cardiac beats, and comparing them with the number of waves on the tracing, the totals were found to agree; the feeble beats were not appreciable to the finger.

October 2nd.—Digitalis in five-drop doses with brandy was given every two hours, but in the course of the day vomiting occurred, and became somewhat urgent; the digitalis was consequently stopped. Vomiting, however, continued, and oral alimentation was suspended, and rectal feeding resorted to.

October 3rd.—For the past two days observations have been made with a view to ascertaining whether the periods of asphyxia were associated in any way with Cheyne-Stokes breathing. But although it was frequently observed that during the period when no pulse was appreciable at the radial, the breathing was very shallow, and that at the onset of the convulsions the respiration became very noisy, nothing like genuine Cheyne-Stokes breathing was observed. On auscultation over the cardiac area to-day, it was observed that during the powerful contractions the sounds were quite audible over the left ventricle, or at least over the point of maximum cardiac impulse; and that over the pulmonic area the second sound was markedly reduplicated, whereas during the feeble contractions the sounds were scarcely, if at all, audible over the left ventricle; but on approaching the pulmonic area they again became distinctly, although very distantly, audible, and here also no reduplication of the second sound could be distinguished.

On carrying the stethoscope up from the pulmonic area to the region of the second right costal cartilage, only one feeble sound was heard; and so far as could be determined by means of a differential stethoscope, this was the first sound. To-day all vomiting has ceased, and oral alimentation has been resumed.

October 4th.—The pulse to-day has become quite regular and of excellent tension and volume. The patient expresses himself as feeling much better, and he sleeps at frequent intervals. The pulse-rate to-day is 72.

October 5th.—Some irregularities were again noted to-day, and the “pulsus bigeminus” has been recorded. The pulse rate is 64.

October 6th.—The tracing taken to-day shows the “pulsus bigeminus” to be well marked. Some irregularities similar to those noted on October 5th were recorded.

October 9th.—Pulse is quite regular, numbering 72 per minute. On auscultation over the heart, the first sound is heard to have a murmurish termination, and the second sound is reduplicated over the pulmonic area. The apex impulse is visible and tangible in the fifth inter-space, three quarters of an inch to the inner side of the nipple line. Respirations number 20 per minute. There is nothing in the condition of the lungs calling for note. The urine contains no albumen and no sugar.

October 10th.—The patient was dismissed from hospital to-day, and consequently regular observation of the case was discontinued, but the patient was seen from time to time, when the pulse was perfectly regular, and for several weeks he remained fairly well, having been warned to avoid over-exertion.

December 3rd.—The patient was seen to-day, when he complained of the old symptoms having returned some three days ago. He looks ill, his face is pallid, the lips are cyanosed, the eyes sunken, and the tongue is coated with a whitish fur, constipation has been the rule of late, the temperature is normal. He has had a slight cough, but this has been unaccompanied by any expectoration. He had had some threatenings of convulsive seizures, that is, he had felt faint, his face had become pale, but he had not lost consciousness.

These attacks had been of very frequent occurrence. The pulse is irregular, as appreciable to the finger at the wrist, and very slow, but the intervals between the beats do not as a rule last longer than ten seconds. The rate of the pulse varies from 15 to 24 per minute. As a rule there are, however, periods during which the pulse numbers 44 per minute at the wrist. Simultaneous cardiac auscultation detected quite distinct though very feeble cardiac contractions, equal in number to the powerful systoles. Auscultation over the apex impulse, which was situated in the fifth space, half an inch to the outer side of the vertical nipple line, during the powerful cardiac contractions, both sounds were muffled and leathery in quality, but no definite murmur was audible. Over the pulmonic area the second sound was doubled, and accentuated as to its second element. Over the aortic area the second sound was much less pronounced. During the feeble cardiac contractions the point over which the heart's sounds were heard with greatest intensity was at mid-sternum, on a level with the fourth inter-space and fifth rib. The sounds were also heard, but less distinctly, over a triangular area which had for its boundaries three lines joining the following points: The fourth sterno-costal articulation on the left side, the fifth sterno-costal articulation on the right side, and a point at mid-sternum on a level with the sixth rib. Over the whole of this area both heart's sounds (during the feeble systoles) were clearly audible, the second element being the more distinct at the upper angle of the area. On carrying the stethoscope upwards towards the second right costal cartilage the sounds were still heard, but much less distinctly. Also, on listening over the fifth inter-space (where the cardiac impulse was visible during the powerful systoles), there was much diminution of intensity of the feeble sounds.

January 5th.—To-day the patient complained of pain in the epigastrium, breathlessness on exertion, and cough, which was accompanied by a clear expectoration. On examining the abdomen the dulness in the hepatic region was found to measure $5\frac{1}{2}$ inches in the middle line and $6\frac{3}{4}$ inches in mamillary line. Pressure over this area elicited an expression of pain. There was some slight impairment of percussion sound

over the extreme base of the right lung behind. Some fine rales were audible over both bases posteriorly. The urine was turbid, and contained a slight amount of albumen.

January 18th.—There is evident improvement so far as the rhythm of the powerful beat is concerned, no very long periods elapsing between the powerful beats. For the most part the rate has been about 33, the variations being from 26 to 36 per minute. The impairment of the breath sounds and diminished percussion resonance at the right base is rather more marked. The hepatic dulness now measures 7 inches in the nipple line and $5\frac{1}{2}$ at mid-sternum. On auscultation over the precordium, in addition to the 36 powerful cardiac contractions there were a similar number of feeble systoles; both powerful and feeble systoles were quite regular in point of time. The pulse rate was 36.

February 1st.—The patient took exercise to-day, but on returning home he was rather exhausted; the pulse was found to be extremely irregular, at times 5 or 6 seconds elapsing between the beats. During these intervals feeble cardiac systoles were audible, varying in number from 2 to 6.

February 7th.—The pulse is still irregular, but shows a tendency to improvement. There is still very marked pallor of the face and cyanosis of the lips. The measurements of the area of hepatic dulness remain as at last note. The dulness on percussion and enfeeblement of breath sounds over the base of the right lung behind remains much as previously noted.

February 10th.—To day the pulse is noted to be of a very variable rate, sometimes numbering 18 per minute, at others perfectly regular at 33. Cardiac auscultation, however, reveals the fact that the heart beats number from 90 to 100 per minute, showing an average of 3 or 4 feeble beats to each powerful systole. There is some increase in the extent of the dulness of the right lung behind. An exploratory puncture was made over the area of dulness at the extreme base, but no fluid was obtained, the needle apparently penetrating dense tissue. In addition to the facts already described as existing in the cardiac condition, there is observed to-day a murmur, soft and blowing in quality and systolic in rhythm, heard generally over the precordium; over the lower end of the

sternum the murmur has a tendency to a coarser and rougher quality.

March 26th.—The pulse to-day numbers 48 per minute, the cardiac contractions numbering 96. The systolic murmur observed on February 10th is not so marked. Percussion dulness of the right lung behind now extends as high as the lower angle of the scapula. On auscultation over this region the main features noted are deficiency of breath sounds with distant tubularity. Vocal resonance and fremitus are decreased, and there is lessened movement of the right side of the chest during inspiration.

July 1st, 1895.—The pulse rate is 33 per minute, with one or two feeble systoles interposed between each powerful cardiac contraction; shortness of breath on exertion has been urgent. The hepatic dulness still measures $7\frac{1}{2}$ inches in the vertical nipple line, the condition at the base of the right lung indicates improvement, there is now only slight impairment of percussion resonance, and breath sounds are merely deficient. The cardiac murmur has now disappeared, and the facial pallor is replaced by a fairly healthy colour. He has had a tendency to faintness on only two occasions recently.

October 1st, 1895.—The pulse numbers 33 and is quite regular. The cardiac contractions number 66, indicating one feeble systole to each powerful contraction. Beyond this fact the cardiac condition presents nothing calling for note. There have been no seizures since last note, nor any tendency to faintness. Dyspnoea has been present for the most part. The area of hepatic dulness is the same as at last note, and the condition at the base of the right lung remains as before. The urine is free from albumen. The patient is now able to do some light work. He is now able, with some reliance, to count his pulse, that is so far as the powerful beats are concerned, and he states that it has never been lower than 33 to the minute for the last three months. He still complains of a sense of tightness in the epigastric and right hypochondriac regions; his appetite is good, and there has been no tendency to vomit.

The subsequent history of the case is not known, as the patient died in the course of a few weeks and no autopsy was obtained.

CASE II. *Recorded by Stokes.*

Synopsis:—Repeated apoplectic attacks, absence of paralysis, remarkable slowness of pulse. Fatty degeneration of both ventricles.

The patient was 68 years of age and had been for a long time the subject of breathlessness. Stupor and a tendency to sleep were characteristics of the condition which had followed an apoplectic attack about three days previously. Breathing was irregular and the pulse numbered 30 per minute. During the seven years previously he had had twenty so-called apoplectic attacks. Before these attacks he was heavy and lethargic, with loss of memory. When they attacked him his pulse would become even slower than usual. He recovered from these attacks without any paralysis. Oedema of the feet developed and dyspnoea became urgent. Later he was suddenly seized with an attack and died in the course of a few hours. The autopsy discovered the substance of the brain to be of a yellowish colour and somewhat watery. The lateral ventricles contained some fluid, but did not seem to be dilated, although the communicating foramen was enlarged. The lungs presented nothing of importance. Some fluid was found in the anterior mediastinum. The coats of the carotid and middle arteries of the dura were white and opaque from deposition of calcareous material. The right auricle was much dilated. The wall of the right ventricle was the seat of marked fatty degeneration. The left ventricle was also much degenerated and very thin. The aorta was the seat of calcareous deposit. No valvular defect was observed.

CASE III. *By Stokes.*

Synopsis:—Repeated pseudo-apoplectic attacks, not followed by paralysis; slow pulse with valvular murmur.

The patient was a man 60 years of age, who had enjoyed good health until three years previously, when he was suddenly seized with a fainting fit, which would have caused him to fall had he not been supported. These attacks occurred several times in the day and seemed to leave no ill effects. Since that time

he had never been free from such attacks for any length of time, and had experienced at least fifty similar seizures. They are uncertain as to their period of invasion and very irregular as to their intensity, some being very much milder and of shorter duration than others. He was never convulsed and there was never any frothing at the mouth. The duration of the attack is seldom more than four or five minutes, sometimes less. During that time he is quite unconscious.

Cardiac condition.—The impulse is slow, and of a dull and prolonged heaving action. First sound is accompanied by a soft murmur. The second sound is also slightly imperfect. The pulse rate is 28 per minute, prolonged and sluggish in character. The urine is free from albumen, with a specific gravity 1010. Later: Pulse varied from 28 to 30 per minute. There have been two threatenings of fits since admission, both occurring in bed, and which have been warded off by a peculiar manoeuvre. As soon as the patient perceives symptoms of the approaching attack he turns on to his hands and knees, and by keeping his head low he says he often averts what otherwise would end in an attack. In addition to the regular cardiac contractions occasional semi-beats are observed, very feeble, and unattended by impulse, and corresponding to a similar state of the pulse, which thus probably amounts to 36, the evident beats numbering 28. Later: Occasional abortive attempts at contraction were observed, probably amounting to about four per minute. They do not destroy the regular intervals between the stronger sounds, but are heard as it were filling up the spaces. A corresponding state of the pulse could not be recognised.

The patient again came under observation three months later, when the following facts were noted: There was a remarkable pulsation in the jugular vein. The number of reflex pulsations was difficult to establish, but it was more than double the number of the ventricular contractions. About every third pulsation was strong and sudden, the other waves being less distinct. These probably corresponded with the contractions already noticed in the heart. The patient had had scarcely any cardiac attacks in the intervals, but he had experienced premonitory sensations.

CASE IV. *By Burnett.**Case of Epilepsy with remarkable slowness of pulse.*

The patient was a naval officer, aged 46, who had had a single attack of epilepsy 16 years previously. Had a second attack in August 1820. In January 1821 he had five paroxysms in the course of half an hour. These attacks were epileptiform in character. Four months later the paroxysms were slighter and less frequent. He experienced a sense of uneasiness in the epigastrium and dyspnoea. The pulse numbered 36 per minute. On the following day the pulse numbered 20 as a rule, and ranged to 32. During the following week the pulse rate was varied between 28 and 56. There were no seizures during this time.

July 2nd. (Two months later).—Four attacks occurred. The pulse then numbered 56.

July 3rd.—Pulse 24, face very shallow, great tenderness on pressure over hepatic region.

July 9th.—Several attacks, pulse 52.

July 16th.—Frequent slight attacks, epigastric uneasiness continues.

July 17th.—Pulse rate 18.

July 20th.—Several severe paroxysms; pulse rate 14.

July 22nd.—Paroxysms of more frequent occurrence and of greater severity. During the paroxysms the pulse is altogether suspended, face becomes pale and convulsed, and a transient flush then succeeds. The pulse is again felt, and patient regains consciousness, to be again attacked; the pulse beats 74 a minute for perhaps the space of a minute, then it intermits for 7, 8, or 10 seconds. Later in the day the attacks continued and the pulse rate was 20 per minute.

July 24th.—The pulse varies from 16 to 18. Attacks are frequent.

July 25th.—Attacks are frequent, but more like spasmodic twitchings than epilepsy. The pulse is often suspended for 10 or 12 seconds.

August 2nd.—Twitchings continue, but no severe attacks have occurred. Pulse rate 24 a minute. The subsequent

history of the case is unknown beyond the fact that he developed anasarcaous swellings.

Burnett makes reference to a case reported by Morgagni, in which a condition of slow pulse with epileptic attacks is described in a priest of 66 years of age, another feature being pain in the right hypochondriac region. Morgagni does not mention the rate of the pulse, but Burnett infers from a quotation from Gerbezius that it was not below 24 per minute.

CASE V. By Holberton.

The patient was a gentleman, aged 64, who was thrown from his horse and sustained an injury to his neck in December 1834. He experienced pain and stiffness in the neck, he could not rotate the head, and was unable to move in bed. He recovered from the effect of the injury to a great extent; difficulty in moving the head continued for a considerable time. Beyond this nothing of note occurred in the progress of the case for some two years.

In January, whilst out walking, he had a fainting fit. The medical attendant found the pulse beating at the rate of about 20 per minute. A few months later he had a second attack. Holberton first saw the patient in March 1837, when the pulse numbered 33 per minute. This rate was readily altered by certain conditions—notably mental excitement increased the rate, which was followed by a slowing and often by a fainting fit. The general character of the pulse when the patient felt well was full, free, sometimes regular, at other times intermittent. Any disorder of the functions readily affected the pulse and determined the tendency to faint. The attacks increased in frequency as time advanced, and in June 1838 an alarming succession of fits occurred and continued at intervals of from 1 to 15 minutes. The pulse rate fell from 15 or 10 in the minute, which was usual, to 12, 10, 9, or 8, and on several occasions when the patient was quite sensible it was as low as $7\frac{1}{2}$. If the finger were placed on the radial artery the approach of a fit might be known sometimes for a second or two before it manifested itself by any change in the countenance. The pulse would cease before the syncope took

place, and the fit would continue until the heart beat again, when the face would redden and consciousness return with a wild stare and occasionally a snorting and a convulsive action of the muscles of the mouth and face.

The frequency of the attacks was uncertain, sometimes the patient would have two or three in a day; at others only one in two or three days; again perhaps only one in a week or in a fortnight or in a month. Sometimes the fit would be severe and all consciousness lost, at other times there would be a mere threatening of giddiness. He had two particularly severe attacks, one six months before death, the other terminating fatally.

Autopsy.—The *lungs* were healthy. The *heart* was large; the left ventricle was rather thin; the aortic valve was healthy; the right auriculo-ventricular opening was very large, and admitted the points of four fingers and thumb. The left auriculo-ventricular opening was enlarged, admitting three fingers easily. The lining membrane of both ventricles was thickened.

The cranium was very thin, the dura mater was firmly adherent throughout, the cavity of the arachnoid contained a large quantity of serum. Brain substance was healthy; the medulla oblongata was small in size and firm in consistence. The foramen magnum was altered in shape. The antero-posterior diameter was so much narrowed that it would not admit the little finger. There was a general thickening about the dura mater and ligament covering the posterior body of the axis. The articular surfaces of the atlas were firmly ossified to the occipital bone, and permitted no movement whatever. There was no calcareous deposit in the vascular system. The pneumogastric nerves were large, and the middle cervical ganglion on the right was unusually developed.

CASE VI. *By Tripier.*

Epilepsy and slow pulse with cardiac arrhythmia.

The patient was a commercial traveller, 45 years of age. Previous history indicated convulsions during childhood. For

nearly seven years nervous crises occurred at irregular intervals, but frequently accompanied by a slow pulse. The pulse as a rule did not exceed 60 beats per minute, and at the times of the crises fell to 18, 16, 14, and 12 beats per minute; the maximum slowing coincided with the greatest frequency of the crises. These crises frequently followed each other in rapid succession and were occasionally so slight as not to interrupt conversation, although the patient sometimes lost consciousness momentarily. The radial pulse was 17 per minute and feeble. The cardiac impulse was not appreciable to the hand. On auscultation over the precordium in the region of the nipple the cardiac beats were heard synchronously with the pulse, but distant and indistinct. Nearer the sternum the same sounds were still heard, but in addition more frequent beats became audible, to the number of 84 per minute. These sounds resembled the beating of another heart. The second sound was not appreciable; these (weaker) beatings were more marked on nearing the sternum, where they attained their maximum intensity. They were also heard to the right of the sternum. They are more clearly struck than those heard towards the nipple. There were pulsations in the veins in the neck, synchronous with the frequent beats, and also other beats more slow.

The patient died suddenly, and nothing of importance was found at the autopsy; both the brain and the heart appeared normal, although no microscopical examination of the heart muscle was made.

CASE VII. *By Tripier.*

Epilepsy with slow pulse and coupled rhythm of the heart.

The patient was a woman 53 years of age, and had typhoid fever at 9 years of age and jaundice at 18. Had suffered from dropsy of face and legs at different periods.

Physical condition.—Some oedema of the lower limbs; the heart did not appear to be enlarged. A soft systolic murmur was heard over the precordium, more marked over the second and third left inter-spaces close to the sternum. Lungs

appeared healthy. Urine was scanty, and contained albumen, blood, and renal débris. In a few days after coming under observation she had an attack of faintness with general tonic spasm of the muscles, but unattended by clonic spasm. On the next day pulse numbered 72, of fair tension, but failing; more attacks of syncope and muscular twitchings, which recurred at frequent intervals. Later syncopal crises were accompanied by loss of consciousness and followed rapidly by cyanosis of the face, but no paralysis. The pulse numbered 44 per minute and was regular. Auscultation of the heart detected strong and feeble sounds. Normal systolic and diastolic sounds were followed by weaker and shorter sounds, diastolic and systolic. The strong systolic sounds correspond with the pulse, but the feeble sounds are not associated with any appearance of radial pulse. Sphygmograms do not indicate any beat during the feeble systole; the heart sounds have never the character of separate sounds. It is a question of a double cardiac revolution giving rise to a single arterial pulse. Thus 44 radial pulse beats and 88 cardiac beats are counted per minute. The following is the order of the occurrence of the sounds and their relation to the silences:

- 1st. Strong systolic sound, short silence.
- 2nd. Strong diastolic sound, short silence.
- 3rd. Feeble systolic sound, short silence.
- 4th. Feeble diastolic sound, long interval,

and so on.

The feeble sounds are like the echo of the strong sounds, and audible over the precordium. The systolic murmur was audible only during the powerful contractions. The cardiac impulse was plainly felt during the strong systoles, but doubtfully during the feeble ones. Jugular pulsation was seen during both strong and weak beats. Minor crises were accompanied by passing unconsciousness, fixity of the eyeballs, immobility, and latterly dilation of the pupil.

The progress of the case was attended by gastric disturbance; the patient died with symptoms of general weakness.

Autopsy.—*Brain.* Meninges slightly thickened and adherent at the base. Middle cerebral and basilar arteries atheromatous;

small haemorrhage of the right side of the pons. *Heart* was enlarged, flabby, and presented marked fatty degeneration. Mitral valve was thickened, the orifice being dilated. The left ventricle was enlarged and the walls thickened. The aortic valves were competent. The aorta was atheromatous; the right ventricle and tricuspid valve were dilated. *Lungs, liver, and kidneys* presented nothing of importance.

In addition to the foregoing cases recorded by Tripier that observer has collected a large number of cases, and from these reports he endeavours to demonstrate the relationship of the epileptiform crises to the cardiac disturbance, and more especially the association of these crises with the double and triple cardiac rhythm. But on Tripier's own showing the majority of these cases have not been reported with sufficient exactitude with regard to the points which they are intended to demonstrate; consequently we have contented ourselves with dealing with Tripier's observations and conclusions concerning these cases, which will be discussed later.

CASE VIII. *By St. George Mivart.*

Epileptiform seizures with unusually slow pulse.

The patient was 61 years of age, and had served in the army and police force. There was no history of neurosis in the family; he had never had syphilis nor rheumatism. He was of temperate habits and had enjoyed fair health, except that "he suffered from his heart," which he attributed to hard work and exposure. Palpitation had been frequent and usually occurred at night. The first fit occurred six years prior to his coming under observation. The second attack occurred two years later; stooping and lowering of the head encouraged the attacks. The last seizure occurred the day prior to his being seen; whilst sitting on a chair he fell forward and had three attacks in succession. The seizures were preceded by a buzzing in the head and a feeling of being unable to stand, with giddiness.

Physical condition.—He looked anaemic, the pupils were equal, the urine was normal, the temperature was also normal,

the tongue was coated. The pulse was regular, firm, and full between the beats, which numbered 20 per minute. The heart's impulse was almost imperceptible, there being only a faint movement at the tip of the sternum. Auscultation revealed apparently the presence of one sound synchronous with the pulse, and heard best towards the apex. One month later the pulse numbered 24, a systolic bruit was heard over the heart, and apparently an aortic systolic bruit. At this time the urine contained a slight amount of albumen. Two months later the pulse rate was 24. The patient was never observed during a seizure, and no note of the pulse either prior to or during the attack was recorded.

CASE IX. By Gibbings.

Remarkably slow pulse with epileptiform seizure.

The patient was 66 years of age, and had a well marked arcus senilis. There was no family history of neurosis. He had had no specific disease, and neither gout nor rheumatism. Dyspnoea was a feature. The pulse was 60 and intermittent. The urine was normal; the heart's sounds were normal; respiratory sounds also normal. Six months later the pulse was 44 and regular, and the patient had frequent feelings of faintness. Later, dyspnoea became urgent. The pulse rate fell to 34, but was regular and synchronous with the heart's beats. The pulse at a later period became slower, and frequent epileptiform attacks occurred. During these attacks the face became pale, the pupils dilated, and the eyes fixed, and the pulse imperceptible at the wrist. After a few seconds the pulse returned, feebly, the face then flushed all over, and clonic convulsive tremors ensued lasting for 5 or 6 seconds.

The first indication of an attack was the non-recurrence of the usual pulse beat. Almost directly there was a moaning sound and the face became fixed. The sequence was observed frequently, so that with the finger upon the pulse it was possible to know when the attack was beginning a second or two before the face changed. Later, the pulse fell to 22 per minute, but was full and regular, the heart's beats corre-

sponding in time. No cardiac lesion was discovered. The attacks became frequent and distressing, occurring both on exertion and during rest. The pulse rate continued to fall, in fact until only 12 and 13 beats were counted per minute. Later, there was an improvement in this respect, 30 to 34 beats per minute being noted. He died suddenly in a faint.

Autopsy.—The right pleura contained some fluid. With this exception all the organs were apparently normal. Dr. Moxon performed the sectio, which is a sufficient guarantee that no gross lesion was overlooked.

CASE X. *By Bristowe.*

Slow pulse with epileptiform seizures.

The patient was 31 years of age, and had been a soldier. There was a history of both syphilis and "fever and ague." He had not had rheumatism, nor had he suffered from palpitation or breathlessness until a few days before he was seen, when he had an attack of faintness caused by over-exertion. Later, he had another attack and lost consciousness. The attacks became more frequent, and he was compelled to give up work. Faintness at the chest seems to have been the first symptom of an attack; this was followed by flushing, on the occurrence of which insensibility supervened. The pulse rate as a rule was 26 per minute, and attacks of fits were frequent. These came on at the end of a period during which there was a considerable interval between the successive cardiac beats. While the pulse was at the rate of even one beat in 3 or 4 seconds no fit occurred, but whenever (and only when) the intermission was prolonged to 5 seconds, a fit followed at the end of that period. A loud roaring systolic murmur was audible above and to the right of the apex, and prolonged thence to the base. Later, the fits had been of very frequent occurrence, and it was noted that prior to the onset of the attack the face became pale and then flushed, and whilst insensibility lasted the hands twitched slightly. Occasionally the pulse beat at the rate of 80 per minute, quite regularly for a few beats, then came a period when one or two beats

occurred at the rate of one or two seconds. Later, the attacks had been more frequent, and on listening to the heart its sounds correspond accurately with the pulsations at the wrist, but between all the beats (attended by waves perceptible at the wrist), separated from one another by intervals of between one and four seconds, there are one, two, or three slight impulses of the heart distinctly to be felt, and seen by the imparted movements of the stethoscope. These latter beats are wholly unattended by cardiac sounds or pulsation in the arteries. Apparently the action of the heart is quite regular in its rhythm. These intermediate impulses, felt at the base of the heart, are they due to the action of the auricle or to the hardening of the relaxed but empty ventricle?

CASE XI. By Hodgson.

The patient was 44 years of age, and came under observation supposed to be suffering from cirrhosis of the liver and kidneys. At first the pulse rate was 108 per minute. Later, it fell to 52 and became intermittent, and continued to fall as low as 24 and 28. Pericardial effusion then developed; following this the pulse rose to 36. Two fits were recorded at this time. Six months later he was again seen, suffering from syncope and fits.

When the pulse stopped for 15 seconds the patient became faint, but when the pause lasted 30 seconds epileptiform spasms ensued. The attacks varied in frequency and severity—sometimes many hours elapsed without the occurrence of a crisis; at other times the seizures and syncopes were of almost constant occurrence, frequently only a few seconds elapsing between two attacks. As a rule the pulse rate was about 21 per minute. On one occasion, however, it was noted to have fallen to 6 per minute. Sphygmograms were taken to show the length of the pause and the fall of the blood pressure. Auscultation and tracings show that the heart was in a state of a systole at the time of the pause. The pulse rate never rose above 36. A gumma of the rib was discovered. The subsequent history of the case was not given.

CASE XII. By Jones and Clinch.

The patient was a man, aged 60 years, who had been an inmate of an asylum for some ten years. He appears to have been addicted to alcohol, and he was also the probable subject of syphilis. After a residence in the asylum of ten years he was suddenly taken ill whilst at a meal with a syncopal attack. Slowness of the pulse was noted, the rate varying between 40-26-20-11 beats a minute. Breathing was laboured. This state was followed by a series of epileptiform attacks, and it was noted that the time of onset of the fits was preceded by a considerable interval between the pulse beats. On one occasion fifteen seconds elapsed between two beats. During one minute eight beats were counted on auscultation. During the fit, the expression was of mental anguish, with protrusion of eyes and dilated pupils. Flushing of the malar regions was marked. The respirations were noisy, slow, and laboured. Subsequently to a fit, the pulse rate increased to 50-60 per minute. The fits varied in intensity and extent—sometimes only slight unilateral facial twitching, at others the convulsion was general. Physical examination revealed: The cardiac dulness was increased both to the right and left, and on auscultation an occasional hard presystolic murmur was detected. The second aortic sound was sharp. The pulse was slow, large, collapsing, and of low tension. Degeneration of the arterial coats was very marked. Lungs—right, deficient resonance from the apex to the base, and medium-sized rales on auscultation. Examination of the nervous system detected no evidence of paresis. The right knee jerk was absent, the left was present; ankle clonus was elicited on both sides. The patient lived for two weeks after the onset of the first convulsion, and a period of six days elapsed without the occurrence of an attack. The pulse varied between 21 and 58. A few days later engorgement of the right lung increased. Right ptosis and contractions of the right occipito-frontalis muscle were noted. Laryngo-pharyngeal palsy became evident. Conjugate deviation of the head and eyes to the right, and some jerking of the right

sterno-mastoid; alternate flushing and palsy of the face, and slight obliteration of the right naso-labial fold were all features of the case leading up to a fatal issue. The necropsy revealed softening and anaemia of the brain substance generally. The basal vessels were not markedly diseased. The heart was large, dilated, and the walls had undergone fatty degeneration. The valvular orifices were dilated and the mitral and aortic valves were slightly thickened. The aorta was dilated, the lungs were congested and oedematous, and old, scattered, tubercles were detected. The left kidney was markedly caseous. The spinal cord was small, and the second right sacral ganglion was converted into a cyst, and a small tumour was found in the third sacral root.

CASE XIII.

Pugin Thornton reports the case of a woman, aged 30, who had had tracheotomy performed for syphilis of the upper air passages in 1872. At that time the pulse rate was 40, and six weeks later it had fallen to 16-24 a minute. It appeared that the patient had been under the care of Dr. Ransome of Nottingham, in 1870, and his notes of the case are very interesting and descriptive. The patient had attacks of an epileptiform nature, associated with a very slow rate of the pulse, the average rate being about 24 beats per minute. The attacks lasted for a period of two months. A severe fit began with sudden pallor of the face, complete loss of consciousness and motor power, cessation of the heart's action for several seconds, on one occasion for 18 seconds. Respiration then became quickened and almost stertorous, the face flushed, the eyes suffused, fixed and turned upwards. She foamed at the mouth. After a time consciousness returned, the expression became calm, and no signs of distress remained; the intellect became clear, and the pulse returned to the normal rate of 24 per minute. Hallucinations were frequent. The intensity of the fits varied—sometimes twelve occurred in a quarter of an hour—and were of a transient nature, and the patient could take up the thread of conversation which had been interrupted by the attack.

Wilkinson reports a case of a man, aet. 62, with a pulse rate of 26-38, with suppression of alternate beats, some of the second beats being palpable, and seen on the sphygmogram. The patient occasionally lost consciousness, but had no fits and no spasms; the attacks are said to have lasted five minutes. The patient frequently fell on the back of his head.

The earliest record which we have been able to find of this type of disease is that described by Burnett in 1825. There can be little doubt that this was clearly a case of inhibited cardiac action immediately preceding the occurrence of epileptiform convulsions; the sequence of events being failure of the pulse, as appreciable to the finger at the wrist, pallor of the face, onset of convulsions, and then apparently simultaneously a subsidence of the convulsion with reappearance of the pulse, flushing of the face, and return to consciousness; the intervals which elapsed between the stoppage of the pulse and the onset of the convulsions varying in length from 10 to 15 seconds. In the uncertainty of their occurrence, and in the varying degrees in the severity of the attacks this case closely resembles the one already recorded; another point of similarity being that at times the pulse beat at the rate of 74 per minute and quite regularly, and then suddenly there was a complete suspension for many seconds; a fact of some importance also being that sometimes the attacks were confined to slight spasmodic twitchings.

Holberton's case is equally clearly one of epileptiform crises following the temporary suspension of the pulse. The observation that an impending attack might be predicted by keeping the finger on the pulse when the latter was lost for a second or two prior to the commencement of the convulsion—the pallor of the face being succeeded by the flushing and the simultaneous reappearance of the pulse, and the return to consciousness with a wild stare—is almost identical with the facts as observed in Case I. Another point of resemblance was the uncertainty of the time of occurrence and the variability in the degree of severity of the crises.

Of the two cases reported by Stokes, although both appear to be cases similar to those of Holberton and Burnett, neither is

reported with sufficient exactitude to allow of any definite opinion being formed; notably no information is afforded as to the state of the pulse immediately prior to the occurrence of the seizures. In one of these cases the so-called pseudo-apoplectic seizures were probably of the nature of epileptiform spasms accompanied by loss of consciousness, more especially as such seizures were of frequent occurrence and left no paralytic sequelae. The other case is interesting, for here there is a condition in which a pulse of 28 to 30 per minute obtains (with more or less feeble cardiac contractions interposed), and in which during the attacks consciousness was frequently lost, but at no time was convulsive spasm observed. In this particular the case was characterised by the occurrence of the minor degree of the critical manifestations, many of which were observed by Holberton, Burnett, and the writer. The manner in which the patient was frequently able to ward off impending seizures by elevating the trunk and lowering the head points very strongly in the direction of the insufficient supply of arterial blood to the brain as being the cause of the attacks. The statement that the attacks never lasted longer than 5 minutes is ambiguous, and probably during such a lengthened period the patient was rapidly passing from one syncopal attack into another.

St. George Mivart's case is apparently one in which a slow rate of pulse existed, with or without feeble cardiac systoles being interposed, and was associated with faintness and epileptiform manifestations. The fact that this patient was never seen during an attack by the recorder of the case deprives this report of much of its value, as we have no information as to whether there was any lengthened suspension of the pulse prior to the attacks. The observation that the pulse which beat at the rate of 20 per minute was full and firm between the beats is interesting, and is quite contrary to the facts noted in the present case. Gibbing's case so closely resembles those of Holberton and Burnett that it is scarcely necessary to do more than indicate this fact. One point, however, in this record is the fact that the patient generally experienced an aura which consisted in the sensation as if hot fluid were trickling down the neck.

Considerable interest and importance attach to the report of Bristowe's case, more especially as that observer lays great stress on the fact that, while the pulse was beating at the rate of 1 beat in 3 or 4 seconds, no fit occurred; but that whenever, and only when, the intermission was prolonged to 5 seconds an epileptiform seizure followed at the end of that period. It would appear, however, that with such short periods of suspension of the pulse many of these attacks were of the nature of minor epileptiform manifestations.

Hodgson's case is scarcely of less importance and interest, and is in striking contrast to that reported by Bristowe, for here such periods as 15 seconds of pulse suspension were merely associated with faintness, and epileptiform attacks only supervened after such lengthened periods of pulselessness as 30 seconds. This case, in some ways perhaps, more closely resembles our own than do any of the others. Notably it presented these exceptionally long periods of asphyxia; another point of similarity being that on occasions the seizures occurred in such rapid succession that only a second or two elapsed between two attacks. In this case also we have the first mention of the fall in the blood pressure being indicated on the sphygmographic tracing.

Jones and Clinch's case is interesting in that it is accompanied by sphygmographic evidence, which is conspicuous by its absence from most of the other records. Here we note also that considerable intervals, sometimes as long as 15 seconds, elapsed between pulse beats, and the lowest rate of cardiac contraction was eight beats per minute (by auscultation). On some occasions, however, the pulse rate rose to 60 per minute. The variation in intensity and extent of the convulsive attacks is very notable.

Pugin Thornton and Ransome's case is remarkable for the similarity of the description of the convulsive attacks to those of Holberton and Burnett.

The case reported by Day was another of those in which very long periods of asphyxia preceded the convulsive attacks and the crescendo reappearance of the pulse on the return to consciousness.

Before referring to Tripier's cases it will be here convenient

to give a brief description of the tracings accompanying this paper, as it will be necessary to refer to them in discussing Tripier's observations.

Tracing No. 1 (September 23rd, 1894) was taken soon after the patient first came under observation, and indicates the termination of a period during which the pulse was quite regular, three of such beats being registered. Then follows a period during which the pulse is apparently absolutely in abeyance. These periods of asphyxia were of variable duration—sometimes 10 or 12 seconds, at others only 4 or 5 seconds elapsing between the pulse beats, this state of matters lasting for 30 or 40 seconds; then a number of more powerful beats are registered, increasing in force in a crescendo manner, to be again succeeded by a series of beats at intervals of 4 or 5 seconds. During the greater part of the time when this tracing was being taken the patient lay quite still. Facial pallor was very marked, and although consciousness was not lost and no convulsive spasm was observed, a certain degree of mental inactivity or stupor was evident.

Tracing No. 2 indicates three long periods of asphyxia, separated by short intervals, during which more or less powerful beats are recorded. The return of the pulse in a crescendo manner is well demonstrated. During these long pauses the facial pallor gradually increased until it became almost death-like. Unconsciousness gradually supervened, and towards the end of the pause epileptiform spasm ensued, commencing in the muscles of the face, but being confined to the head and neck; the return to consciousness being heralded by the return of the pulse and the coincident flushing of the face.

Tracing No. 3 presents a combination of the characters exhibited by the two preceding ones, the periods of asphyxia varying in length in a most marked manner.

Tracing No. 4 shows a pulse of excellent tension and of perfect regularity.

Tracing No. 5 indicates also a pulse of good tension, but here and there it exhibits a tendency to the character of the "pulsus bigeminus."

Tracing No. 6 demonstrates the bigeminal character in a more perfect manner.

Tracing No. 7 indicates a reversion to the type seen in Nos. 1, 2, and 3.

Tracing No. 8 shows a general pulse rate of approximately 20 per minute, but with numerous small waves recorded on the down stroke, or diastolic part of the tracing. These waves vary greatly in number, and correspond in point of time with the occurrence of feeble cardiac sounds, which were audible on auscultation over the precordium.

Tracing No. 9 also demonstrates the small waves on the descending line.

Tracing No. 10, practically similar to No. 9.

Tracing No. 11 in its first part indicates the frequent occurrence of long pauses. Following the third beat there is a gradual fall of the down stroke (indicating a fall of blood pressure). In the first part of the pause, and notably in that part which is above the level of the commencement of the previous percussion stroke, we find small waves registered; these, however, disappear when the blood pressure falls to a certain level. The second half of *Tracing No. 11* belongs to an earlier date, September 27th (No. 7), and is included, as it shows the crescendo return of the pulse, with the bigeminal arrangement of the beats after a period of asphyxia.

Tracings Nos. 12, 13, and 14 show pulse beating for a time quite regularly (see No. 14), with a sudden stop and fall of the blood pressure, the pause in one case (No. 14) lasting for nearly 30 seconds, and the record of succeeding beats being distorted by the convulsive action, together with the startled movement of the patient, on the return to consciousness. Convulsions ensued at the latter end of each of the long pauses registered in these tracings, which were the only ones we were able to obtain showing the very long periods of asphyxia and consequent convulsions, as in most cases when the convulsion was very severe the tracings were destroyed by the instrument being displaced from the artery by the spasmodic movements. These tracings were not taken with the view of demonstrating the small waves, but rather to show the length of the pause and the fall of blood pressure.

Tracings Nos. 15, 16, 17, 18, and 19 show the return to regularity and the improvement in tension which occurred

and was maintained until the patient was dismissed from hospital. The modification at the end of tracings Nos. 15 and 18 was adopted to demonstrate the tension by exaggerating the tidal and dicrotic waves.

The remainder of the sphygmograms serve to indicate the varying character of the pulse, both in volume and in rhythm, over a period of many months. Some of them also demonstrate points of importance, which will be referred to in due course.

Although the two cases which Tripier observed himself were reported with the view of supporting the theory that the epileptiform seizures preceded and were the cause of the cardiac arrhythmia, it has been necessary to include them in this paper in order that we might examine Tripier's reasons for formulating his opinions. In both of these cases we have a condition in which a slow pulse obtained, and this was associated with epileptiform spasms.

Of the other cases which have been collected by Tripier we do not propose to deal in detail. An extract taken from his remarks on these cases will be sufficient to indicate his views. Concerning one case he says:—"On ne saurait douter que le malade ait eu des crises épileptiformes coïncidant avec des troubles cardiaques et notamment avec l'arrêt du coeur et la production d'un rythme couplé. Ce dernier phénomène est signalé dans l'observation avant les crises, de telle sorte qu'on pourrait croire que celles-ci étaient survenues consécutivement et même qu'elles dépendaient du trouble cardiaque, surtout d'après la manière dont l'observation est rédigée. Mais nous rejetons cette interprétation en nous basant sur toutes les observations analogues rapportés précédemment, dans lesquelles nous voyons les crises épileptiques ou syncopales précéder évidemment les déviations du rythme cardiaque, tandis qu'aucune observation antérieure précise ne permet d'admettre le contraire."

And again:—"Du moment où l'attention n'était pas particulièrement appelée sur les crises, celles-ci ont pu passer inaperçues au début."

And lastly:—"Il n'est guère admissible que les phénomènes convulsifs à répétition, avec perte de connaissance, sans souffrance et sans le souvenir des accidents éprouvés, puissent être

attribués à une lésion du cœur, à une endocardite, fût-elle très évidente, ce qui n'était pas le cas, puisque les valvules se présentaient dans les conditions où on les observe ordinairement avec une hypertrophie aussi considérable du cœur.

“Au contraire, tout s'explique parfaitement avec les crises épileptiformes, à caractères variables et atténués, plus ou moins fréquentes, subintrantes à certains moments, comme on les observe du reste avec les déviations du rythme cardiaque.”

Among the records of the cases which form the basis of these observations by Tripier we find that now famous and frequently quoted one of Holberton. Tripier, while he admits that the facts as recorded by Holberton point to the cardiac failure as preceding the onset of the convulsions, denies the validity of the argument that the cardiac failure was the cause of the following convulsions.

It would appear that nothing could be more explicit than Holberton's description of the pulse failure immediately preceding the convulsion and the subsidence of the latter on the reappearance of the pulse, and we therefore cannot accept Tripier's interpretation of the facts. It certainly would appear unwarrantable to assume that slight epileptiform spasms may have occurred before the pulse failed, and that these minor manifestations were overlooked. As has been previously pointed out, Tripier, in his own records, gives no indication that the pulse was regular before the spasm occurred. Unfortunately, Holberton's case is unaccompanied by any sphygmograms, but in the collection which accompanies this article there are two tracings, one of which, we think, may explain Tripier's views, without in any way invalidating the opposite theory, and the second unquestionably disproves Tripier's statements. In the first of these tracings, numbered 2 in the collection, we have demonstrated three periods of asphyxia, or pauses in the cardiac action (here we make no allusion to any interposed feeble beats, as none are recorded in the tracing, and we propose to revert to this point later); each of these pauses is followed by a series of beats to the number of 7 or 8. The convulsion supervened during the latter part of the pause and was terminating during the occurrence of the earlier beats of this series of 7 or 8; consequently, if the patient were observed, and the pulse

were palpated at this time, only some 3 or 4 beats would be appreciable prior to the cessation of the pulse and the onset of the next syncopal pause. And it would not be an unnatural inference that the convulsion stood to the cardiac failure as cause to effect. But the fact that the convulsion supervened during the period in which the heart's action was in abeyance almost conclusively proves that the cardiac failure was the cause of the convulsion.

If we now refer to Tracing No. 14 we find that at the commencement of this tracing a series of twelve regular beats are recorded; this is followed by the suspension of the pulse for a period of twenty or twenty-five seconds, and during the later part of the pause (that is after a period of some fifteen seconds of asphyxia) the convulsion supervened, to terminate on the recurrence of the pulse. If Tripier's view be correct, a convulsion must have occurred to cause this cardiac stoppage. Such a long pause in the cardiac action as is here indicated would surely have necessitated the occurrence of a very violent convulsive spasm, and it is highly improbable that such a convulsion, or indeed a minor seizure, would have occurred without being observed, or without interfering with the manipulation of the sphygmograph, more especially as we had the advantage of knowing Tripier's views on the subject, and we were naturally careful that even very slight epileptiform manifestations should not escape observation at such a juncture.

It has been pointed out that the convulsion did not terminate immediately the pulse reappeared at the wrist, the explanation of this being that after such a lengthened period of asphyxia as twenty seconds the brain was in such a profound state of anaemia that several cardiac contractions were necessary to re-establish the cerebral arterial circulation to such a degree as to allow of the brain again resuming its functions.

Further, the return to consciousness and subsidence of the convulsion were invariably heralded by this return of the pulse and the flushing replacing the facial pallor.

With regard to Tripier's statement in one of his reports that the greatest frequency of occurrence of the convulsive seizures was coincident with the maximum slowing of the pulse, the following facts may be noted: Tripier makes no reference to

this slowing being accompanied by irregularity. The lowest rate of pulse recorded by him is 12 beats per minute, and still convulsive seizures occurred. We must assume that in Tripier's case the pulse was regular, since he does not furnish us with any evidence to the contrary.

We have now reached one of the most important points in the whole question of the relationship of cardiac derangement to convulsions. The foregoing records have almost conclusively proved that when cerebral anaemia is the cause of the convulsion, the cardiac disability has preceded both. Tripier's views differ entirely from those of the majority of observers with regard to cause and effect, but he is not alone in the view that the greatest frequency of the convulsions coincides with the maximum slowing of the pulse. Broadbent believes that the convulsion is the result of the cerebral anaemia dependent upon the cardiac slowing, as the following quotation abundantly shows :

"The epileptiform attacks are not often violent, but resemble petit mal rather than a typical epileptic fit; while, however, the convulsion may not be so severe, there is profound unconsciousness, not like epileptic coma, but of a syncopal character, and the pulse may be extremely infrequent, sometimes less than 20 in the minute. In my judgment the heart failure as manifested by the slow pulse, and the consequent arrest of the cerebral circulation, are the cause of the fits, and it is not the epileptiform attack that affects the action of the heart."

We see here, then, that while Broadbent differs from Tripier very decidedly with regard to cause and effect, both observers lay much stress upon cardiac slowing associated with convulsions. Balfour, who has had a most extensive experience of bradycardia, tells us that Hope believed that "when one or two beats are regularly and permanently imperceptible in the pulse, such cases contribute the bulk of these cases in which the pulse is described by auscultators as being singularly slow, as, for instance, 30 or 40 per minute." In a few rare cases, however, it is really slow. Balfour's experience would point in the opposite direction. He says: "So far as my experience goes, the rarity has been all the other way, as

I have seen many more really slow hearts than hearts beating at the normal rate with an abnormally slow pulse, due to alternate hemi-systoles." There can be little doubt, therefore, that these two varieties of so-called bradycardia, real and apparent, do exist.

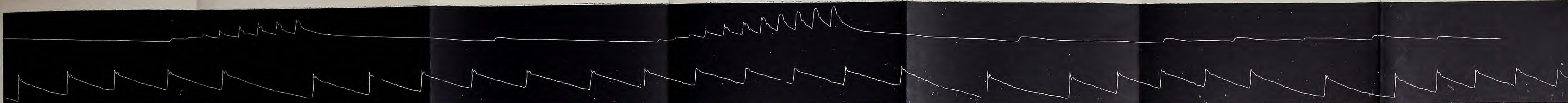
Describing the true form of bradycardia, Balfour says: "As a rule the steady, slow, funereal beat never varies from the time of its commencement until the patient's death." Of the other form he describes the case of an elderly lady whose radial pulse numbered only 20 beats per minute, but whose cardiac contractions were 60 per minute. This lady was the subject of frequent epileptiform seizures. Referring to Holberton's case, he prefaces some remarks on the syncopal attacks by the statement that, although several of his patients had died during a syncopal attack, he had never seen such a seizure. From this we gather that he has never seen the cessation of the pulse for some seconds prior to the onset of convulsion, as described by Holberton. Balfour here raises the most vital point in the whole question. He clearly points out that regularity of the heart's action is a cardinal character of bradycardia, and yet he has never seen syncopal, and probably epileptiform, crises associated with true bradycardia. Dr. Hewan, who was an accurate observer, had a slow pulse habitually, the rate varying from 24 to 32 beats per minute. Yet he never experienced syncopal attacks or convulsions of any kind, and he was able to indulge in such severe physical exercise as mountain climbing. Burnett is very explicit in his statement that sometimes the pulse rate was over 70 per minute, and yet the patient became convulsed. Holberton found the pulse rate as low as eight beats per minute, and yet perfect consciousness was maintained, and presumably he means also that no convulsive seizure occurred. It is interesting to note the frequency with which slow pulse, or bradycardia, or infrequent pulse are described in association with epilepsy in the literature of the past 20 years, but it does not appear that cardiac arrhythmia has been considered a causal factor in the production of the convulsion. Tripier has pointed out that cardiac arrhythmia frequently attends convulsive seizures. He, however, considers that the convulsive attack

precedes the arrhythmia. The problem which presents itself is, how does the cerebral anaemia occur, and how does it give rise to convulsions when a slow pulse obtains? Holberton has recorded a pulse rate of eight per minute, without the occurrence of convulsive seizure. Burnett has recorded a pulse rate of over 70, which was associated with convulsion.

If we refer to the tracing No. 50 of the series which accompanies this article, we see that a hitherto perfectly regular pulse, regular both with regard to its force and rhythm, is followed by a syncopal attack and epileptiform seizure. The epileptiform seizure was certainly of a very modified type, as the asphyxial pause was of short duration. Again, the tracing No. 14 presents a perfectly regular character of pulse in its early part, a regularity which had obtained for a considerable period (at least for several minutes) prior to the onset of convulsions. The pulse rate here was something like 30 per minute. We have frequently seen a pulse rate of less than 20 per minute, and this condition obtaining for several hours or days without the occurrence of the convulsive attacks. We are therefore compelled to find some explanation of the cerebral anaemia which produces the convulsion other than that of simple cardiac slowing. A pulse rate of eight per minute is surely sufficiently slow to induce cerebral anaemia and convulsions. Yet Holberton did not note convulsion when such a condition obtained, but he did note, and he recorded the fact, that for several seconds before the onset of the convulsion the pulse was entirely in abeyance. It would therefore appear more probable that it is the cessation or irregularity of the cardiac action for a period which induces the cerebral anaemia, such cessation, to all practical purposes, as is most perfectly indicated in the earlier sphygmograms accompanying the paper. This is a very different state of matters from the slowness of the pulse, or bradycardia, which Balfour assures us is always associated with regularity.

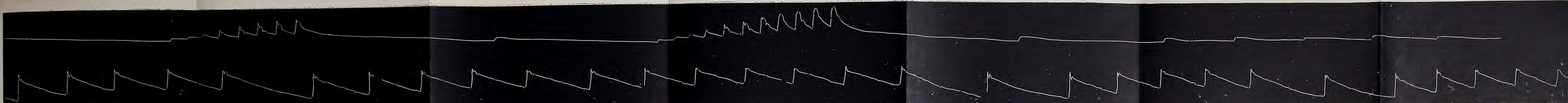
Our own experience, which extends over many hundreds of observations of the case under discussion, would tend to show that a slow rate of pulse of 18 to 24, *e.g.* Nos. 54, 55, 56, where even a moderate degree of irregularity obtains, is not associated with convulsions. That a slow rate of pulse is

1894
Sept. 27
No. 7



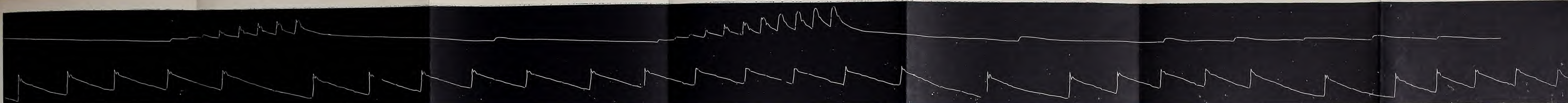
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Sept. 29
No. 8



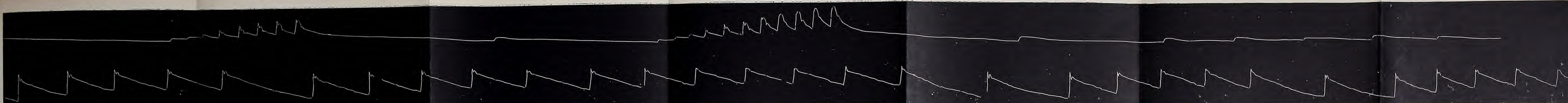
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Sept. 30
No. 9



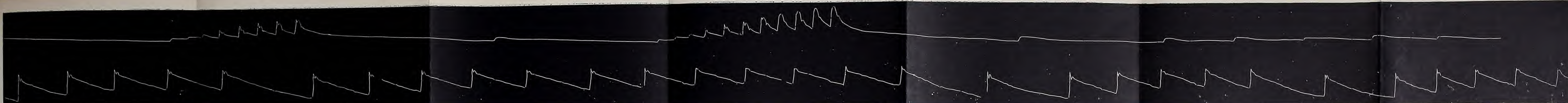
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Sept. 30
No. 10



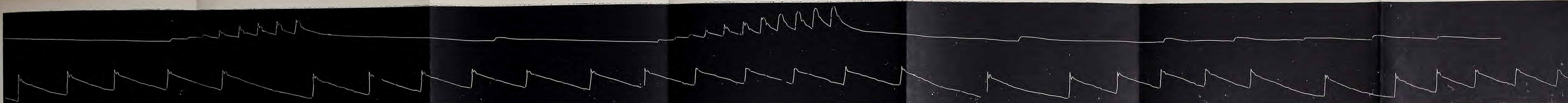
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Oct. 1
No. 11



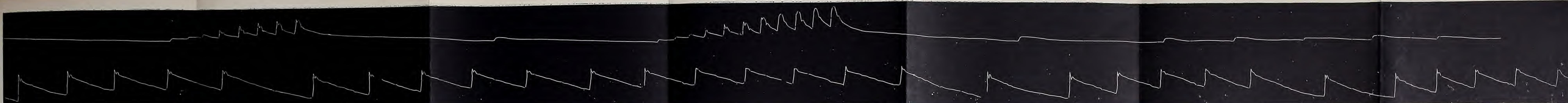
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Oct. 2
No. 12



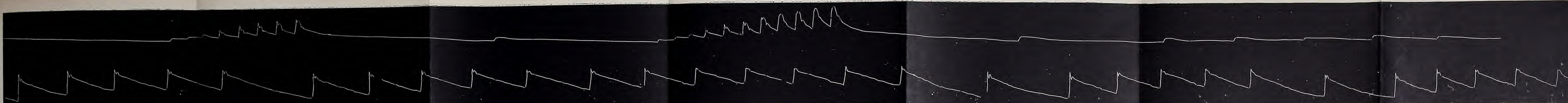
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Oct. 2
No. 13



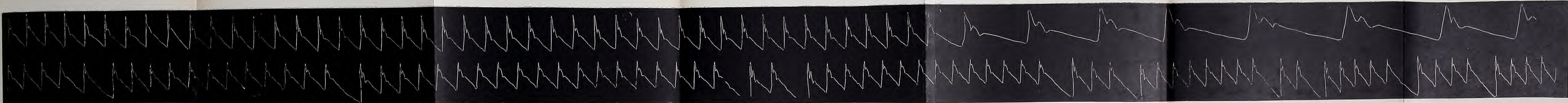
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Oct. 3
No. 14



14

1894
Oct. 5
No. 15



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Oct. 6
No. 16



16

Oct. 6
No. 17



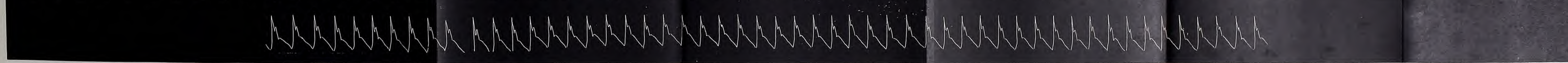
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Oct. 8
No. 18



18

Oct. 9
No. 19



19

1894
Oct. 11
No. 20



20

Oct. 15
No. 21



21

Oct. 20
No. 22



22

Oct. 27
No. 23



23

Nov. 6
No. 24



24

Nov. 9
No. 25



25

Nov. 23
No. 26



26

1894
Dec. 31
No. 27

1895
Jan. 1
No. 28

Jan. 1
No. 29

Jan. 1
No. 30

Jan. 2
No. 31

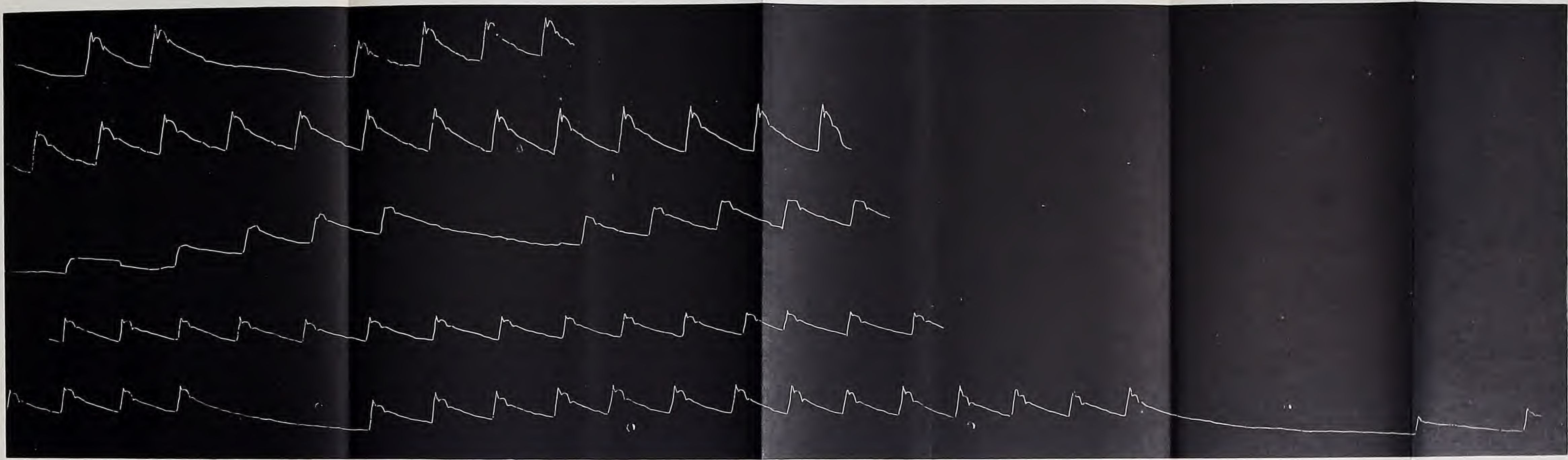
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1895
Jan. 2
No. 32

Jan. 2
No. 33

Jan. 2
No. 34

Jan. 2
No. 35

Jan. 3
No. 36

Jan. 3
No. 37

Jan. 4
No. 38

Jan. 5
No. 39

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1895
Jan. 6
No. 40

Jan. 7
No. 41

Jan. 8
No. 42

Jan. 8
No. 43

Jan. 10
No. 44

Jan. 12
No. 45

Jan. 14
No. 46

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1895
Jan. 27
No. 47



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Jan. 27
No. 48



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Jan. 28
No. 49



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Feb. 2
No. 50



50

Feb. 2
No. 51



51

Feb. 4
No. 52



52

1895
Feb. 14
No. 53

Feb. 15
No. 54

Feb. 17
No. 55

Feb. 18
No. 56

Feb. 19
No. 57

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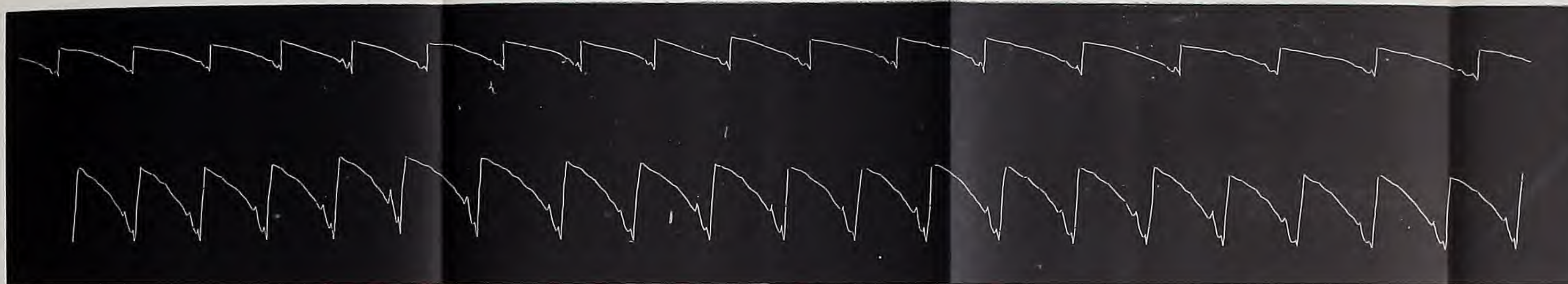
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1895
March 29
No. 58

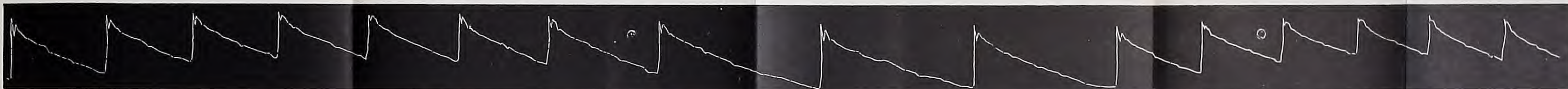


58

May 3
No. 59

59

No. 60



60

often associated with convulsion is fully established, but it is only where such slowing of the cardiac action is associated with irregularity that epileptiform seizures occur. Irregularity may be either in the way of rhythm or of volume of the pulse, but in these cases it is the irregularity in the force of the cardiac contractions, or in the volume of the pulse, rather than in the rhythm, otherwise it would be difficult to understand exactly when the cerebral anaemia occurs. Take for example a pulse rate of 20 per minute, we have not more than a period of three seconds of asphyxia, and three seconds of asphyxia is not sufficient to induce cerebral anaemia in so marked a degree as to cause convulsive spasm. Bristowe noted this in his case. We would even go further and say that with a pulse rate of even 12 per minute, in which we have a period of five seconds of a pause between each pulse beat, provided perfect regularity obtains, convulsions do not supervene. We would imagine that a pulse rate of twelve beats per minute merits the distinction of slow pulse.

In the early stages of the case under consideration, a period of from five to seven seconds of asphyxia induced convulsive seizure, whereas latterly such a length of pause was only associated with, at the most, cerebral inactivity, or stupor, and a period of 10 to 15 seconds was necessary to induce convulsions. And, of course, the more profound the anaemia, or, in other words, the longer the asphyxial pause, the more general did the convulsion become.

So far there has been a consensus of opinion with regard to the cardiac disturbance preceding and causing the convulsion, Tripier being the only observer who believes otherwise.

We have now come to a point on which there is considerable difference of opinion, viz., the question as to whether the cardiac arrhythmia or slow pulse is associated with abortive cardiac systoles, or interposed feeble beats.

Stokes, Bristowe, and Tripier in their reports all describe the occurrence of feeble beats between the powerful systoles. In one of Stokes' cases, at least, there was one feeble systole between each powerful cardiac contraction. Bristowe describes a number of feeble beats, varying from 1 to 3 between each powerful systole.

Tripier also found several, but probably not more than three, feeble systoles interposed between the strong contractions. In the present case, the number of feeble systoles varied from time to time within very wide limits; sometimes only one weak beat was heard, at others as many as 20. In tracing No. 9, for instance, these feeble beats varied in number from 2 to 5.

Broadbent's view is rather different. He believes that the feeble systoles do occur, but he also believes that a slow rate of pulse may exist without their occurrence, and he quotes the cases of St. George Mivart and Gibbings in support of this view, and certainly these were cases in which very slow pulses, of 24 and 12 per minute respectively, existed. We can only say that no feeble systoles were observed. There certainly appears to be no reason why a slow pulse with irregularity, without any interposed feeble systoles, should not be the cause of epileptiform crises; because, if these feeble beats are not appreciable at the radial pulse, they certainly cannot materially affect the cerebral circulation: so that so far as the effect on the cerebral circulation is concerned, these beats are practically non-existent; and we have, therefore, to all intents and purposes, a condition identical with that in which an irregular and slow pulse obtains. In tracing No. 60, these feeble beats are fairly well brought out; the original tracing, however, demonstrates their occurrence to greater perfection.

We take it for granted that no exception will be taken to the statement that these small waves on the downstroke of the tracing do exist in fact. We must, therefore, endeavour to explain their origin. It has been suggested that they were caused by the rhythmical contraction of the arterial wall. The objection to this explanation is the fact that on auscultation over the precordium distinct, although very feeble, cardiac sounds were audible.

It is scarcely necessary to refute the statement that they may have been due to rhythmical tremors. We are satisfied that during all the asphyxial pauses these feeble cardiac systoles were taking place, and that in the accompanying tracing these small waves are the radial expression of cardiac contractions.

Two points in connection with these feeble beats, as expressed by the sphygmogram, are very striking. In the first place it will be noted that they occur at absolutely regular intervals, between the powerful beats, or on what appears to be the diastolic part of the tracing. Secondly, they are wholly unlike the abortive beats seen in a sphygmogram, taken from a case either of fatty degeneration of the myocardium or mitral disease, where we have an overburdened or feeble muscle making ineffectual efforts to contract. In neither of these conditions do we see the absolutely rhythmic contraction of even the feeblest beats, such as we have here recorded. They bear no resemblance to the feeble beats of a *pulsus bigeminus* or a *pulsus trigeminus*, frequently observed after treatment by *digitalis*; although they did occur after the exhibition of *digitalis* they were noted before that drug was administered.

We are now confronted with the reasonable objection that if they were the result of feeble cardiac systoles, why were they not indicated in all tracings, and also why were they not recorded during the whole of the intervals between the powerful systoles? We must here refer to tracing No. 9 (which is almost identical with the tracing No. 60). In it we find these small waves recorded on the downstroke in each interval between the powerful systoles. A careful examination of these tracings will discover the fact that at practically no time does the downstroke fall below the level of the commencement of the preceding upstroke, that is to say below the level of the base line, thereby indicating no great fall of arterial tension; and, therefore, so long as we have such a column of blood in the vessel as is indicated by this tension, so long do we find these beats recorded.

If we now examine tracing No. 11 we see following the third powerful beat a long pause; during the earlier part of this pause, before the downstroke has fallen below the level of the base line, these small waves are recorded, but diminishing in size, and ultimately disappearing after the base line is passed.

This gradual fall of the downstroke necessarily means fall of blood pressure, which is synonymous with emptying of the

vessel, so that at the end of the pause there is practically no blood in the vessel, and consequently the means of communicating these small waves is non-existent. What the mechanism of this profound fall in the blood pressure, or outflow of blood to the periphery, for it could not well have been due to a regurgitation, was, will be discussed later. The second part of the objection that these waves were not indicated in all tracings, as, for example, 1, 2, 3, and 14, may be met by the explanation of the great pressure which it was necessary to exert upon the artery to keep the lever on the recording paper during such alterations of blood pressure. Tracing No. 14 demonstrates our point. The first dozen beats, which are situated at the very top of the paper (which indicates that the tension was at its highest), were sufficiently distorted, and yet the pressure necessary to cause this distortion was barely sufficient to keep the lever on the recording paper at the latter end of the long pause, which indicated the minimum of tension, or the maximum fall of blood pressure. It is not difficult to understand that if this pressure effaced some of the details of the powerful beats, it should also entirely obliterate the smaller waves.

We have endeavoured to make it clear that the cardiac irregularity is the primary factor, and that the epileptiform attacks are caused by the cerebral anaemia, resulting from such suspension of the cardiac action. We have also indicated that we are disinclined to the view that the heart's action was entirely in abeyance during the long pauses such as are indicated in the first three tracings.

Hodgson has given it as his opinion that in his case the heart was in a state of asystole, and he assumes it as proved by the absence of any indication of pulse waves, however small, during the pauses. This may be true, but it is not necessarily evidence that no feeble systoles were taking place, and it is difficult to understand how, with such a fall in the blood pressure as must have occurred in 15 or 30 seconds of pause which Hodgson describes, any pulse wave could be expected to be registered. However, we must keep in view the statement that during this time no feeble sounds were audible over the precordium. We have also pointed out that so far as the

cerebral circulation is affected the presence of these feeble systoles is of no importance, as their existence neither increases nor lessens the cerebral anaemia. With regard to the cardiac condition, however, the presence of these feeble beats is of great importance.

In the present case these feeble systoles were accompanied by sounds which were so difficult of appreciation that they frequently escaped observation, and on one occasion a most experienced and skilful auscultator failed to detect them until the time of their occurrence was indicated by small waves on the sphygmogram, and this notwithstanding the fact that they were believed to be taking place.

Bristowe, Stokes, and Tripier all describe these feeble beats. Broadbent has also observed them. Tripier would seem to indicate that they might be due to right-sided contraction alone; and his description in one case is very similar to our own, when one powerful systole was followed by a single feeble one. With regard to the occurrence of these feeble beats, Bristowe, at the end of the report of his case, makes the following statement:—"Apparently the action of the heart is quite regular in its rhythm; these intermediate impulses felt at the base of the heart, are they due to the action of the auricle or to the contraction of the relaxed and empty ventricle?"

In our own observations, when it was first discovered that these feeble sounds had their point of maximum intensity situated in the region of the sternum, at a level of the fourth, fifth, and sixth ribs, we were inclined to the possibility of their being due to a right-sided contraction.

This idea was supported by the fact, that during the feeble systoles the second sound was not heard over the aortic area, from which we inferred that the aortic valves were not closing, whilst both sounds were audible over the pulmonic area. (With regard to the description of these sounds, Tripier suggests that they seemed to originate in another heart. Our own impression would be that they resembled the muffled ticking of a watch, or they might be likened to the sounds of a slowly-beating foetal heart.) This view, however, was not tenable for obvious reasons, the chief one being that we had demonstrated oscillations in the column of blood in the radial artery. It

was more probable, therefore, that the left ventricle was contracting, and that, if the ventricle contained any blood, it was the propulsion of this blood against the aortic valves being only sufficiently powerful to raise the aortic curtains. And this puffing out of the aortic valves communicated a wave to the column of blood in the aorta.

This view gained some support from the fact that, so far as we could determine, the second sound, during these feeble beats, was not audible on auscultation over the aortic area.

Another explanation which suggested itself was, that the ventricle was contracting, but that it did not contain sufficient blood even to raise the aortic curtains, and that the wave of muscular contraction was carried along the ventricular wall to the aorta, where it set up an oscillation in the blood column. It is difficult to decide which of these two theories explains the origin of the waves, because during the earlier part of the pause (that is to say, when the pauses were of no great length, as, for example, in tracing No. 60) there could not have been much blood in the ventricle, and yet here the waves were as distinct as those in the later part of the pause. Whereas, on the other hand, at the latter part of the pause, the ventricle must have contained a considerable quantity of blood, as, at the following powerful systole, a considerable volume of blood was thrown into the aorta, as indicated on the tracing by the height of the percussion wave.

Whichever of the explanations be accepted, we are now confronted with the difficulty of explaining, first, the fall in the blood pressure; secondly, the absence of the small waves from the tracing during the latter part of the pause. It did not appear probable that the left ventricle was sending any blood into the aorta. Even although some blood were thrown into the aorta during the feeble systoles, this would scarcely have been sufficient to cause the onward passage of the blood, which is indicated by this fall of pressure.

There can be no question but that the sphygmogram No. 14 indicated a fall of blood pressure, and this means an emptying of the blood vessel. This emptying must either have been of the nature of an onflow of blood to the periphery or to a regurgitation towards the heart. That it was not the latter

seems almost certain, as we had no auscultatory evidence of the fact. Neither does it seem probable that there was a condition of stasis in the arteries; for in that case we should have been able to define the rounded outline of the artery, and this was not possible. The only explanation, therefore, is that there was an outflow of blood to the periphery, and that so long as a certain column of blood remained in the ascending part of the aorta to receive impressions, either from the aortic valves or otherwise, so long did we get these feeble contractions indicated on the tracing. The powerful systoles of the ventricle gave sufficient impetus to the column of blood in the aorta to carry it to the periphery; this being all the more possible owing to the blood passing constantly into a wider vascular area, and through the capillaries into the veins, where the resistance to its onflow was not greater than the impetus which it had acquired from the force of the cardiac contraction.

As soon as the powerful systole was over, the vessels would tend to return to a state of rest, and would only oscillate slightly while there was sufficient blood in the aorta to receive the impressions from the feeble systoles, so that the outflow of blood to the periphery was of a passive nature at the latter part of the pause. Although the fact that the second pulmonic sound indicated that the right ventricle was contracting, and the pulmonic valves opening and closing, it would not appear that this contraction was sufficient to drive the blood through the lungs into the left ventricle; the left ventricle was, nevertheless, receiving a supply of blood, as was indicated by the following pulse, and probably the powerful contraction of the right ventricle during the last systole was sufficient to propel the blood through the pulmonary circuit, and, the tension here being much greater than in the left heart, the onflow of blood was insured, although the feeble action of the left auricle could not supply any *vis a fronte*.

Before proceeding to the question of causation of the cardiac inhibition, there are certain points in the progress of the writer's case which may not inaptly be referred to.

The presence of a systolic murmur, audible over the point of maximum intensity of cardiac impulse, was evidently the result of the enfeebled condition of the ventricle, with dilation

of the mitral orifice, and consequent regurgitation. Another murmur of a rougher quality and more superficial in character, and most marked over the lower end of the sternum, was probably due to tricuspid reflux. Both these murmurs disappeared in the course of time, the pulse having improved in tension and the heart having recovered some of its tone. The sense of fulness and uneasiness in the epigastric region, in the latter stages, was associated with a considerable increase in the measurements of the area of hepatic dulness, and was probably dependent upon venous stasis resulting from the cardiac embarrassment. This engorged condition of the liver and consequent congestion of the portal radicals will probably explain the gastric irritability and intolerance. It is interesting to note that this symptom, referable to the liver, was a feature of Burnett's case, and also of the one referred to by that observer, as recorded by Morgagni. The altered condition at the base of the right lung behind was at first thought to be due to the presence of fluid in the pleural sac. Exploratory puncture, however, did not confirm that idea; it was more probably of the nature of a congestive induration of the lung with thickening of the pleura. Its ultimate disappearance did not give support to the view that it might be of malignant origin.

The record of this case is unfortunately incomplete, as no autopsy was obtained. However, as the chief aim has been to discuss the case from a clinical point of view, this loss, though great, is not sufficient to deprive the case of much of its value; and although we cannot throw any light upon the subject with regard to the probable causation of the cardiac inhibition, from our own experience, this paper might be less complete if we made no reference to the opinions of the observers of other similar cases.

The first autopsy that we find recorded is that on Holberton's case. This is a most important report, for there can be no doubt as to the relationship of the cardiac failure to the epileptiform crises. They stand, unquestionably, as cause to effect. But the information obtained at the autopsy opened up many possibilities with regard to causation. The history of injury, and the symptoms referred to the base of the skull and neck, naturally attracted Holberton's attention, as here he

expected to find the cause of the cardiac disturbance. And there can be no doubt that the alterations which were found in the structures around the foramen magnum and the medulla oblongata suggested very strongly that the pressure thus exerted by the inflammatory products had interfered with the functions of the higher cardiac centre. Fortunately, the examination was made in an extremely careful manner, all organs being subjected to scrutiny. The condition of the heart also affords us valuable information. The left ventricle was thin, the lining membrane of both ventricles was thickened, both auricula-ventricular orifices were enlarged. The patient was a man of 64 years of age. Although Holberton does not report any change in the myocardium, a man of 64 years of age with thin-walled and dilated ventricles and thickened endocardium was probably the subject of degeneration of the cardiac muscle.

An autopsy was obtained in one of the cases reported by Stokes, in which very marked fatty degeneration of the heart was found. Of Tripier's two cases the heart was found to be the seat of extreme fatty degeneration in one case; in the other nothing was found to explain the symptoms, although microscopical examination of the heart muscle does not appear to have been made.

In Gibbing's case nothing was discovered in any organ to account for the symptoms. Microscopical examination of the heart muscle was here also apparently omitted.

Jones and Clinch's case presented a complicated problem on post-mortem examination, but there appears to have been well-marked evidence of syphilis of the brain, and probably also of the vagus and spinal accessory nerves. The heart also was the seat of well-marked fatty degeneration affecting the myocardium. The cerebral vessels at the base were not markedly diseased.

We have endeavoured to demonstrate the relationship of the cardiac disability to the occurrence of the convulsive seizures, and by means of the sphygmograph this has been not altogether impossible. But a greater difficulty presents itself in the explanation of the cause of the cardiac arrhythmia, or slowing. According to our present knowledge, the weight of opinion

probably points to some form of degeneration of the myocardium as being responsible for the cardiac disability. In many of the records of post-mortem examinations on such cases we find evidence of fatty degeneration of the heart muscle. Holberton's case was one in point; Jones and Clinch's is another, but in both of these cases there were other factors at work which would readily explain the cardiac phenomena. In the former case there was a condition of the tissues around the medulla, pointing to pressure on the higher cardiac centre or cardiac nerves. In the latter the observers were satisfied that the spinal accessory and vagus nerves were the seat of syphilitic deposit.

Waller questions whether the vagus communicates directly with the cardiac muscle, or indirectly by the intermediation of ganglia; and he says that "Upon the known fact that the action of the vagus diminishes the frequency and force of the beat, the opinion has been based that the vagus terminates in the muscles as well as in the ganglia, diminution of the frequency being regarded as the sign of vagus action upon the ganglia; diminution of force as the sign of vagus action upon the muscle."

Gaskell believes that all parts of the heart are endowed with the power of rhythmical contraction, independently of the cardiac ganglion cells, but varying in degree in different parts; and he thinks that the ganglion cells occupy a purely accidental position, as a result of their former situation along the wall of the longitudinal blood vessel, or in the least modified part thereof. Quain believed that the form of cardiac disturbance under discussion was dependent upon fatty change of the myocardium. But he thought that the mode in which fatty changes affected the pulse depended upon the part of the muscle affected, and upon the extent of the change.

If we examine the tracings numbered from 20 to 23 and from 41 to 46, it would be difficult to believe that such sphygmograms were the radial expression of the contraction of a degenerated myocardium, or at least of a myocardium so degenerated as to exhibit such vagaries as are portrayed in the earlier sphygmograms. In the tracings 41 to 46 many of the characters are those of high tension, and indicate a power-

fully acting ventricle; and during the whole series of the tracings we have not a single example of the abortive systole so frequently seen in the degenerated muscle tracing, as for example in Balfour's work on *The Senile Heart*, Figs. 3 and 4. Balfour does not regard myocardial degeneration as a cause of true bradycardia; and we take it he believes the arrhythmia, or false bradycardia, to depend upon disturbance of the nervous mechanism also.

The testimony of Morrison and Jones and Clinch endorses Balfour's view.

If we examine the tracing No. 60 it is difficult to resist the impression that the heart is acting in response to two kinds of impulses—the one powerful, the other feeble.

These impulses or their expression occur at perfectly regular intervals, although sometimes there are three, at others two feeble beats following each powerful contraction. The problem which presents itself is, where do these two kinds of impulses arise, the one causing powerful and the other the feeble contractions, and why should they have different expressions? Are these impulses being regularly transmitted from the higher cardiac centre, some being augmented in the heart, either in the intrinsic cardiac ganglia or in the muscle itself; or are the feeble beats the result of stimuli originating in the heart itself, and are the powerful beats the result of such stimuli augmented by impulses from the higher centre, but, owing to some abnormal condition either in the cardiac centre or in the cardiac nerves, these impulses do not originate or are not transmitted with their wonted regularity? This is a problem which can be only suggested here, as the object of this paper has been to deal, as far as possible, with the subject in its clinical aspect.

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A SECOND SERIES OF SPECIMENS ILLUSTRATIVE OF CERTAIN CONGENITAL AFFECTIONS OF THE URINARY APPARATUS.

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SOME time ago¹ we published in *The Glasgow Medical Journal* a series of specimens illustrating congenital affections of the urinary apparatus.

Since then we have met with some other examples, and it has seemed to us that the recording of these might prove of value to other observers, not so much from the clinical point of view, but as illustrating certain variations in the development of these organs.

The specimens form four groups, namely:

1. Vascular abnormalities.
2. Cases of diminutive kidney.
3. Varieties of fusion of the kidneys.
4. Cases of hydronephrosis.

¹ February, 1898.

CASE I.

Displacement of Right Kidney, with mobility. Short Renal Vein.

On opening the abdomen in this case the right kidney was at once seen to form a tumour near the middle line. Its lower margin was situated near the middle of the vertebrae, while the proper upper margin was much posterior, the organ lying nearly transversely, the hilum presenting upwards and inwards. In this position it formed a swelling just beneath the lower edge of the liver, immediately outside the level of the gall-bladder. From this position the kidney could be readily displaced forwards till its middle was at the middle line, but it could not be displaced backwards into its normal position. On more particular examination it appeared that the principal cause of its inability to pass backwards to its normal position was the position of the renal vein which seemed to hold the organ upwards and towards the middle line. The renal vein passed into the vena cava 2·5 cm. before the latter entered the liver, the renal vein itself being 3·8 cm. in length. The condition was not discovered during life, and the patient died with cardiac symptoms, apparently related latterly to atheromatous obstruction of the coronary artery. (Western Infirmary, *Path. Reports*, No. 875).

CASE II.

Congenitally small Kidney.

The *kidney* measures, after hardening, 3·5 c.m. vertically, by 2 c.m. transversely at the upper and 1·25 c.m. at the lower end. The convexity is irregular, but on the whole the organ presents the normal reniform outline. The *pelvis*, which is of good size, is situated on the antero-internal aspect of the organ. It measures vertically, where the kidney tissue meets it, 1 c.m. It communicates with the hollowed-out upper extremity of the kidney. The *ureter* passes in the normal direction. The arteries, from a single trunk, pass to the kidney both in front of and behind the pelvis, the posterior

branch turning down towards the lower end of the kidney. Veins correspond generally to the arterial distribution. (Western Infirmary *Path. Reports*, No. 5069).

Microscopic examination showed marked increase in the fibrous tissue of the organ.

The specimen was obtained from a male, aet. 28, who died on the sixteenth day in the course of rheumatic fever. There was a history of intemperance. He had enteric fever twelve years previously. There was accentuation of first sound of heart. The left kidney showed well-marked compensatory hypertrophy.

CASE III.

Atrophied hydronephrotic Kidney on right side; compensatory hypertrophy of left Kidney; double spermatic Artery on both sides. (Fig. 1.)

The drawing of this specimen is reproduced from a sketch by Dr. Robert Fullarton, late demonstrator of anatomy in the University of Glasgow. The condition was found in a dissecting-room subject, and the following description is taken from Dr. Fullarton's notes:

The *left kidney* is considerably larger than normal, and its surface is roughly mapped out in a trilobate conformation.

The *pelvis* and *ureter* present a normal appearance.

The *right kidney* is very small. The upper portion of the *ureter* is dilated and can be followed up into the *pelvis*, which is formed by the union of two calices. These on being opened up are found to extend quite to the periphery of the kidney.

The *suprarenal bodies*, as seen in the figure, occupy their normal position. The left body is of normal size, the right being somewhat larger than usual.

The *vascular arrangement* is unusual:

On the *right* side a small renal artery arises from the aorta some distance below the origin of the superior mesenteric vessel. The branches of this artery enter the upper part of the kidney on the inner part of its anterior surface. In addition the vessel supplies two branches to the suprarenal body.

On the *left* side two renal arteries arise close together from

the aorta at a lower level than the vessel on the right. The upper divides into branches before reaching the hilum, and while the larger of these branches enters the hilum one courses over the front of the kidney to its convexity, where it gives an ascending twig to the left suprarenal body and a descending

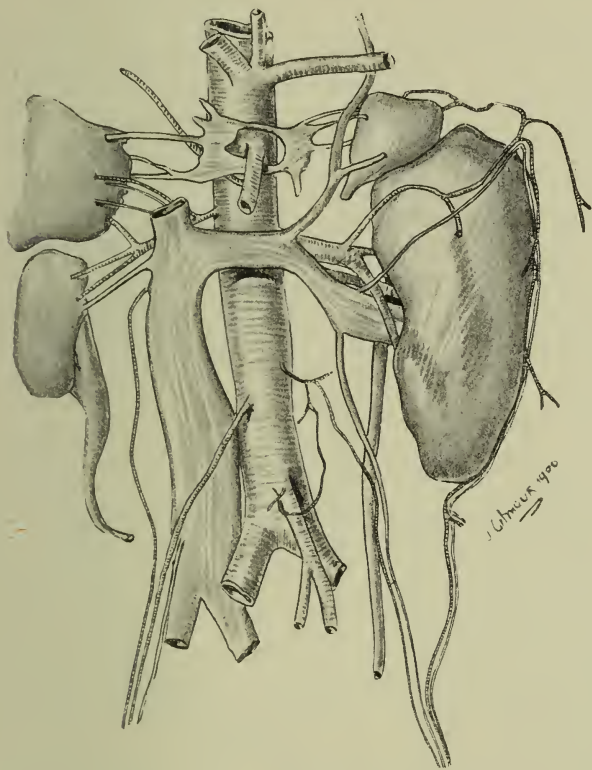


FIG. 1.

one which runs down to the spermatic cord. During this course, branches to the renal substance are given off.

Spermatic arteries; right.—A vessel arising in the usual situation from the aorta is accompanied by a second very slender artery which arises from the front of the aorta behind the left renal vein.

Left.—A small artery is seen having the usual relations. This vessel gives off numerous branches, one of which

anastomoses with a small axis-like trunk which springs from the front of the inferior mesenteric artery. The left spermatic artery is accompanied down the cord by the vessel already mentioned as arising from the left renal.

Cremasteric and deferential arteries were present on both sides.

Veins; right.—One small vein passes from the kidney to the vena cava. Two separate tributaries drain the suprarenal body. A single spermatic vein joins the vena cava a little above the commencement of the latter.

Left.—A comparatively large renal vein drains the left kidney. Joining it from below is the normal spermatic vein, while from above comes the second spermatic vein with a circuitous course round the convexity of the kidney. A large vein comes down past the suprarenal body, which it drains and then empties into the renal vein close to where the latter crosses the aorta.

CASE IV.

Fusion of Kidneys, on the right side of the body. (Fig. 2.)

There was no kidney on the left side, while the right kidney was represented by a large organ having two ureters. This organ, which was of general reniform outline, measured 15.5 cm. vertically by 6.5 cm. transversely at the upper, and 9 cm. at the lower end.

The upper part of the kidney is smooth, contrasting with the lower half, in which lobulation is present even on the mesial aspect.

There are two *hila*, the upper being separated from the lower by a ridge of renal tissue which extends upwards and inwards to the inner margin of the mass and which measures vertically 1 cm. The *upper hilum* is of oval shape with its long axis oblique (upwards and inwards), and is situated antero-internally. The upper pelvis, measuring vertically 4.5 cm., has its apex directed downwards and slightly outwards. The junction of its calices is indistinctly seen. Its wall is hypertrophied, as also is that of its ureter, which passes to the right side of the trigone. The *lower hilum* is situated on the

anterior surface of the broad lower half of the organ. The formation of the lower pelvis from distinct calices is well

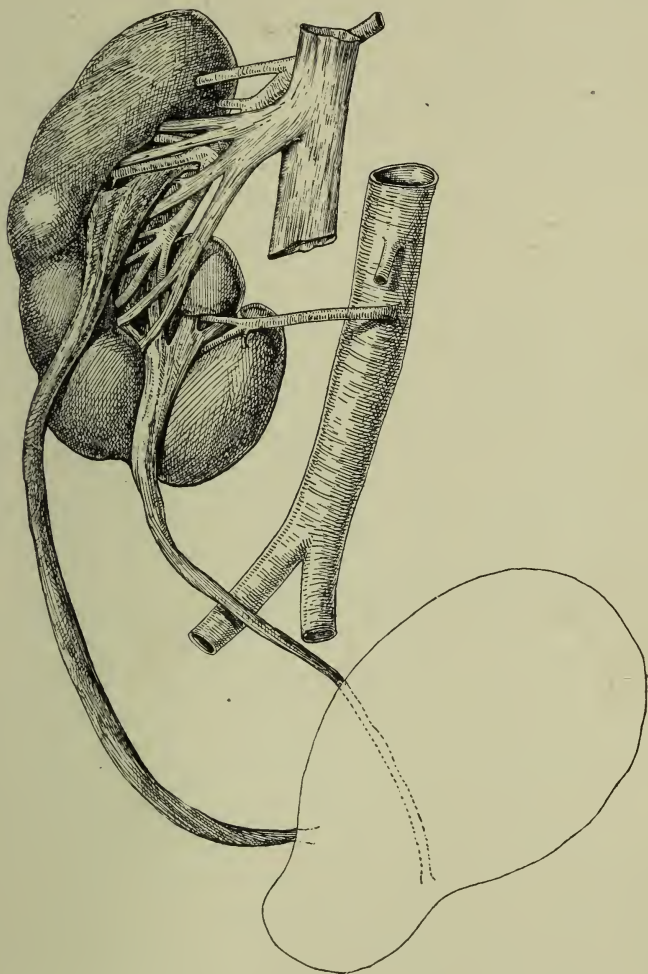


FIG. 2.

shown, five of these being seen, separated above by wide intervals. The pelvis passes downwards in a groove between the lobules at the lower end of the kidney, and the ureter passes to the left side of the trigone.

As regards thickness of wall, both pelvis and ureter of the

lower part of the organ contrast markedly with the corresponding structures from the upper part.

Vessels.—A large artery (whose connection with the aorta has not been preserved in the specimen) descends to supply the upper part of the organ. Before reaching the hilum it gives off two branches of considerable size, each entering a depression on the inner surface of the kidney. Of the branches in the hilum, two at the upper end pass outwards in front of the pelvis and lie in front of companion veins (these latter, however, at their junction lie anterior to the trunk of the artery). A third branch of artery passes outwards behind the pelvis, at the upper end of the hilum. At the lower part of the hilum, two branches pass outwards behind the pelvis, and from this part of the main vessel two branches pass down to the lower hilum, one externally, the other to the inner side of that cavity. The lower part of this hilum receives a large branch from the aorta, arising 1 cm. below and to the left of the inferior mesenteric artery. This vessel supplies two branches to the kidney substance before it enters the hilum. The veins from both hila correspond to the arteries, but enter the vena cava by a single trunk on the right side.

The following is a summary of the notes made at the *post-mortem* examination: Male, aet. 69.

The lower ureter crossed the right common iliac artery a little more than 2.5 cm. below the aortic bifurcation and the proximal portion of the artery was slightly dilated. The wall of the ureter was thin and almost translucent. The right ureter was much thickened and hyperæmic, and contained grumous material similar to that in the bladder. The fundus of this organ was occupied by loose dendritic masses of soft tumour-tissue, encrusted with phosphates. The orifices of the ureters were patent, admitting a probe 2 mm. in diameter. The prostate was normal. A smooth-walled multilocular cyst was present in the peripheral portion of the kidney surface, in the neighbourhood of the lower calices of the upper hilum. In the pyramidal tissue just above this was a small irregular cavity whose walls exhibited hæmorrhagic infiltration.

Microscopic examination showed cancer of the bladder. There was evidence of an ascending septic infection extending along

the right ureter to the upper part of the kidney. (Western Infirmary, *Path. Reports*, No. 5907.)

Note.—This specimen is undoubtedly formed by a fusion of the left kidney with the lower end of the right.

This misplaced left kidney presents the discoid appearance which is often found in cases where the kidney lies in an abnormal situation (see former series, Figs. 1 and 2, *Glasgow Medical Journal*, vol. i., 1898) and it contrasts with the reniform outline of the upper half of the mass (see also Fig. 10 former series), which represents the right kidney.

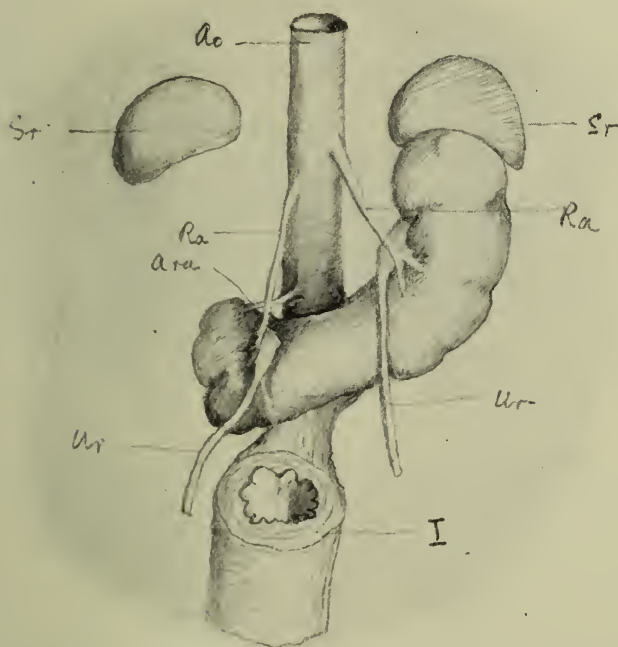


FIG. 3.—References in the figure : *Ao*, Aorta ; *Sr, Sr*, Suprarenal bodies ; *Ur, Ur*, Ureters ; *Ra, Ra*, Renal arteries ; *Ara*, Accessory renal artery ; *I*, Intestine, cut across.

CASE V.

Fusion of Kidneys in horse-shoe-like form in a human embryo at about the 10th week (Dr Gemmill's case). (Fig. 3.)

A full description of this case appeared in the *Glasgow Medical Journal*, January, 1900, p. 55, but it has seemed to us

that it might be of some interest to draw attention to the following points:

The arrangement of parts might be considered as a condition intermediate between the regular "horse-shoe" and the so-called "sigmoid" kidney. The origins of the arteries correspond to what we have already described in Case IX. of our first series¹ in so far as the lowest vessel supplies the upper end of the right kidney; this would be the inferior extremity of a proper "sigmoid" kidney. The drawing of which Fig. 3 is a reproduction was made by Dr. Gemmill by reconstruction from serial sections.

CASE VI.

Horse-shoe Kidney; ill-developed pelvis in left half. (Fig. 4.)

The *kidneys* are nearly equal in size, the right measuring 10·7 cm. by 8·8 cm., the left, 12·25 cm. by 9·5 cm. They are united at their lower extremities by an isthmus of renal tissue measuring 3·8 cm. vertically. This is nearly 1·25 cm. thick at its lower part, but for 2 cm. above it is marked by a deep vertical furrow, both in front and behind, so that the bridge is here not more than ·3 cm. antero-posteriorly. Lobulation is not present. In both kidneys the *hilum* is situated antero-internally. The *pelvis* occupies the greater part of the vertical extent of the hilum in the right and is of normal dimensions, while in the left the individual calices are well seen, and a pelvis, as such, hardly exists. Three calices from the upper part unite halfway down the hilum, and the resulting tube is further added to, at the lower margin of the depression, by two calices, an outer and an inner.

The *ureters*, which are normal in appearance, pass down in distinct grooves on the anterior surface of their respective kidneys.

The *vascular relations* have not been preserved, but what remains of them shows that, in the *right* three arterial branches lie in the posterior wall of the hilum. These are arranged in vertical series; the upper passes in front of and the middle goes behind the pelvis, while the lower running outwards behind and below the origin of the ureter lies in front of

the pelvis at its lower end. About the middle of the outer lip of the hilum is seen another arterial branch passing outwards in front of the pelvis (not shown in the figure). No veins are preserved.

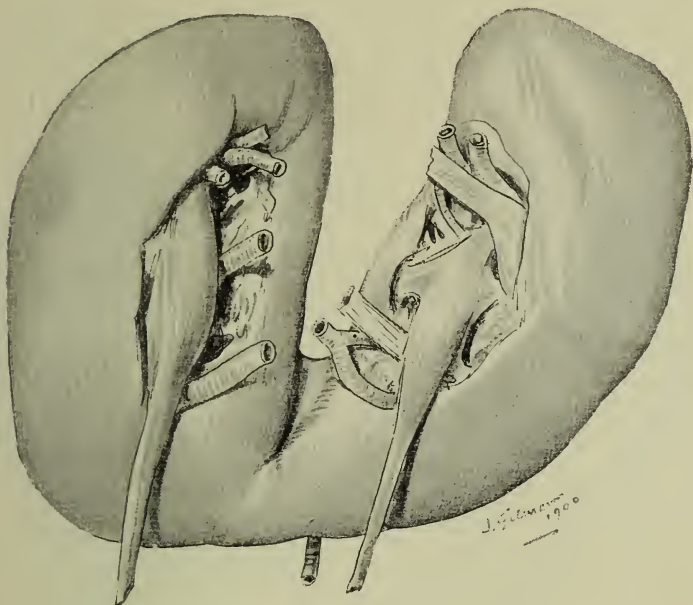


FIG. 4.

Left.—In the upper end of the hilum two large arteries are found branching one in front of the other behind the upper calyx. In front of the former is seen a large vein. At the lower end of the hilum a large artery bifurcates, the upper branch passing behind the tubes representing the pelvis, while the lower passes out under the arch of the ureter and in front of the lower calices. Companion veins are found here. *Posteriorly* an artery enters the isthmus from below, and two veins emerge from the extremities of the furrow.

The above specimen is from the Pathological Museum of the University College, Dundee. Clinical particulars are unfortunately wanting.

CASE VII.

Rupture of Horse-shoe Kidney ; extensive haemorrhage ; rupture of left renal vein. (Fig. 5.)

The kidneys have been united at their lower extremities by an isthmus measuring 4·7 cm. from above downwards, and 1·7 cm. from before backwards. This has been torn across

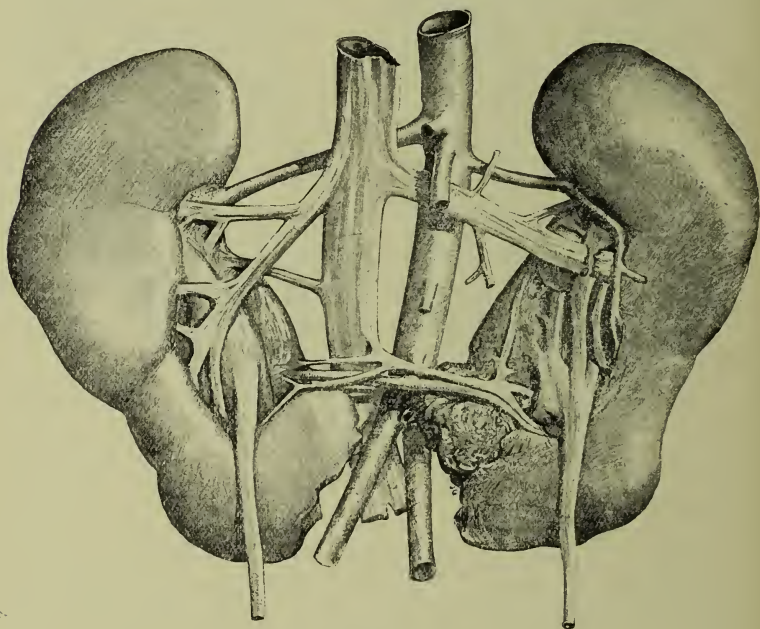


FIG. 5.

somewhat obliquely in a vertical direction so that the rough torn surface of the left kidney looks forwards while that of the right looks mainly backwards. The torn surfaces were separated by a distance of 5 cm. one from another.

The kidneys measure from above downwards, right 14 cm., left 14·5 cm. While their posterior aspect is smooth, their anterior surface presents slight lobulation, most manifest in the lower part of the right kidney.

The *hilum* is antero-internal in the right kidney and anterior

in the left; from the respective pelves the ureters descend in grooves on the anterior surface of the corresponding kidneys.

The *right pelvis* is long and narrow and is joined by four tubes. The *ureter* leaves it on its anterior surface about 1.5 cm. above the lower end and passes downwards with an outward direction. The *left pelvis* is much smaller and is formed by the junction of six tubes. The *ureter* arises just above the lower end and has a downward and outward direction.

Vascular supply: arteries.—Each kidney receives two branches from the aorta, a right upper branch arising from its lateral aspect slightly above the level of the origin of the superior mesenteric artery and a left upper branch arising slightly below the origin of that vessel, also from the lateral aspect of the aorta. Lower branches pass outwards above the isthmus from a common trunk arising 2 cm. above the bifurcation of the aorta.

Veins.—On the *right side* an upper renal vein joins the vena cava at the ordinary level. It receives branches from the whole of the outer border of the hilum. From the lower end of the hilum two branches pass to join the lower renal vein of the *left side*. There is also a vein coming from the middle of the inner lip of the right hilum and emptying into the vena cava 2 cm. below the right renal vein.

On the *left side* the inner margin of the lower end of the hilum is drained by the lower renal vein, already mentioned, which empties into the vena cava 6.5 cm. above the junction of the common iliac veins. From the outer lip of the hilum an upper renal vein proceeds and opens into the vena cava opposite the right upper renal vein, crossing the aorta to do so. This vein was found at the autopsy torn across near its origin at the hilum.

The aorta and vena cava pass downwards behind the renal isthmus.

Microscopic examination of both kidneys showed no evidence of any pathological condition.

History.—Male, aet. 33, fell into a ship's hold, a distance of 14 feet. Great abdominal tenderness, especially in hypogastrium; bloody urine drawn off by catheter; ultimately

resistance in left flank, great pain and desire to micturate; lived for 48 hours. During last 14 hours of life, only a few drops of bloody urine obtained by catheter. *Post-mortem*: rupture of small intestine 60 cm. from pylorus, and another 65 cm. lower down; both opposite mesenteric attachment. Large retroperitoneal clot enveloped the kidneys. Genitals were normal. (Western Infirmary, *Path. Reports*, No. 5416.)

This is apparently a unique case. We have been unable to find any record of a case of this kind in renal literature.

CASE VIII.

Horse-shoe Kidney, with unusual vascular relations. (Fig. 6.)

The kidneys are united at their lower extremities by an isthmus of renal tissue measuring 3.5 cm. from above downwards, and a little more than 1 cm. from before backwards. The isthmus lies at the bottom of an oblique groove running downwards from left to right.

The kidneys measure from above downwards, the right nearly 13 cm., the left, 12.5 cm. The posterior aspect is smooth, but anteriorly lobulation is present, markedly in the right, and to a lesser extent in the left. The lower lobe of the right kidney is almost separated from the rest of the organ by a fissure situated externally.

The *hilum* in the right kidney is antero-internal above and internal below, while in the left kidney it is internal and even looks slightly backwards.

The *right pelvis* is very short, measuring about 1.25 cm. long by 1.5 cm. broad at its base, and is formed by the union of five separate tubes. It is situated at the lower end of the hilum, and the upper tubes are correspondingly longer than the lower. The *ureter* leaves the pelvis at its lower end and passes downwards and outwards on the surface of the lowest lobe of the kidney, 2 cm. from its outer margin.

The *left pelvis* is longer than the right, and measures 2 cm. in breadth and in length. It is roughly triangular in outline, and receives four or five tubes from the hilum, at the lower end of which it is situated. The *ureter* arises at its lower

extremity and passes downwards and inwards over the lowest renal lobe, 6 cm. from its outer border.

Vascular supply.—The *right kidney* receives two branches arising from the aorta, one in front of the other, about 5 mm. below the origin of the superior mesenteric artery. The

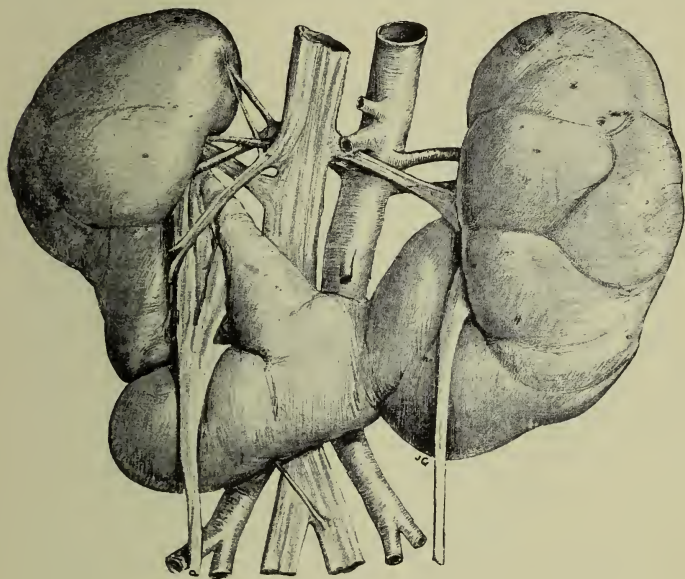


FIG. 6.

anterior passes to the upper end of the hilum, while the posterior divides into two—an upper branch to the upper end of the kidney proper, and a lower to the upper half of the hilum.

The fissure separating the lower lobe from the convex outer margin of the kidney contains an artery coming from behind and supplying the lower half of the hilum. This vessel, which is not shown in the figure (Fig. 6), springs from the right common iliac, 2 cm. below the bifurcation of the aorta.

The *left kidney* receives two branches from the aorta—an upper arising opposite the corresponding vessel on the right side, and a lower, 1 cm. further down. The latter branch is distributed mainly behind, and the former in front of the pelvis.

Veins.—The *right* kidney has an upper renal vein passing transversely to the vena cava from the upper end of the hilum. Entering the vena cava immediately above this vessel is a long vein which courses up in front of the pelvis from the deep fissure at the lower lobe, and which receives branches along the anterior lip of the hilum. This vein at its entrance into the vena cava is joined by a twig from the upper end of the kidney substance. A branch from the posterior aspect of the lower lobe, near the convex margin of the kidney, passes to the *left* common iliac vein.

The *left* kidney is drained by one large vein whose tributaries are found both in front of and behind the pelvis.

Microscopic examination of both kidneys showed normal appearances.

The specimen was obtained from the body of a youth 19 years of age. It occupied the middle line, the lower edge of the isthmus being situated 2 cm. above the sacral promontory. No other malformations were found. The cause of death was a wound of the heart.

CASE IX.

Horse-shoe Kidney; dilatation of pelvis. (Fig. 7.)

The long axes of the kidneys form an angle of about 45° , at the apex of which the organs are joined at their narrow lower extremities by an isthmus of renal tissue, which is flattened antero-posteriorly and measures .5 cm. in this direction by 3.75 cm. vertically. The inferior mesenteric artery passes down in a shallow groove in front of the isthmus, while behind is the front of the left half of the aorta. The *kidneys* are slightly lobulated in their lower parts. The *right* measures 11.5 cm. vertically by 5.5 cm. at its upper and 6 cm. at its lower extremity. The lower end curves upwards and inwards and lies in front and to the right of the aorta, while behind the latter and further to the right is the vena cava.

The *hilum*, narrower above than below, is situated antero-internally and contains the proximal portion of the dilated *pelvis*, which in its distal part overlaps the inner margin of the

kidney (*i.e.* posterior lip of hilum) by slightly over 2 cm. The *ureter* comes off from the lower end of the anterior surface of the pelvis, with which it forms an acute angle. Its origin is situated 5 cm. above the lower margin of the hilum, and the duct passes over the front of the kidney in a groove between the lower two lobules.

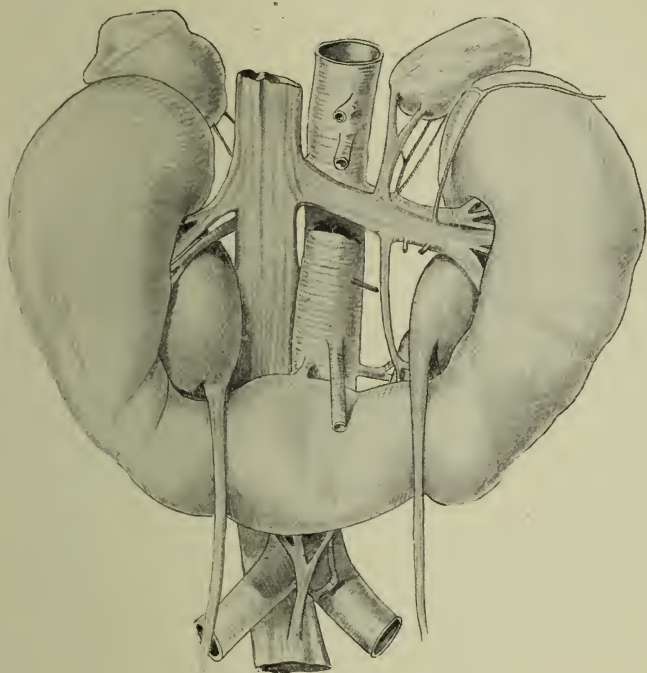


FIG. 7.

The *left kidney* measures 12·5 cm. in length by 6 cm. transversely at the upper and 5 cm. at the lower end. The posterior lip of the *hilum* is very prominent below, but this is not the case above, the hilum here being situated more on the inner aspect of the kidney. The *pelvis*, while not so much dilated as that on the right side, projects inwards beyond the posterior lip of the hilum for a distance of 1 cm. The *ureter* arises on its inner aspect at a distance of about 1·5 cm. above the lower end of the hilum. It forms an acute angle

with the pelvis, and passes downwards in a groove on the anterior surface of the lower end of the kidney.

Vascular arrangement.—The *arteries* are in three sets: 1. About 1.25 cm. below the origin of the superior mesenteric artery a renal vessel arises from either side of the aorta. That on the right is slightly lower than the vessel on the left and passes out behind the right renal vein to the upper end of the hilum in front of and above the pelvis. The left has the same distribution and course. The right suprarenal artery arises directly from the aorta above the corresponding renal vessel, while the left suprarenal springs from the left renal artery, 1 cm. from its origin.

2. About 1 cm. below the origin of the inferior mesenteric two branches are given off from the antero-lateral aspects of the aorta. Of these the right passes outwards in a fissure on the posterior surface of the kidney and enters the lower end of the hilum posteriorly to the pelvis. The left enters the lower end of the corresponding hilum, which it supplies, sending a branch upwards and outwards behind the pelvis.

3. From the bifurcation of the aorta, on its inferior aspect springs a vessel which runs upwards in front of the parent trunk to enter the lower margin of the isthmus in the middle line posteriorly.

The *veins* correspond to the arteries, save that No. 2 on the right joins the vena cava, while on the left it passes up and joins the renal vein internal to the spermatic vein; it receives tributaries from the left ureter. No. 3 has additional tributaries from the right side of the isthmus and empties into the termination of the left common iliac on its anterior surface.

The *suprarenal veins* fall into corresponding renal vessels. In addition, the left renal receives a communication from a large vein running along the convexity of the upper and outer part of the kidney and partly connected with the inferior surface of the suprarenal body. This may possibly be a supernumerary spermatic vein. (Cf. Case III., Fig. 1, p. 465). The clinical history was unfortunately not obtained.

CASE X.

Horse-shoe Kidney; double hydronephrosis; calculus in right pelvis. (Fig. 8.)

The kidneys, very unequal in size, measure, the right 10·6 cm. from above downwards by 4·3 cm. transversely, the left 12·8 cm. by 6·25 cm.

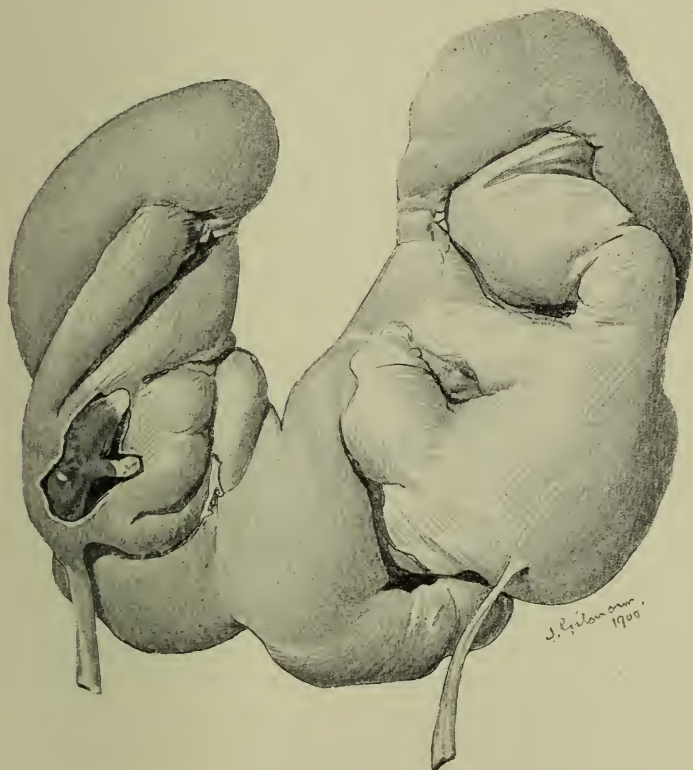


FIG. 8.

They are united at their lower extremities, not by any distinct bridge but as if fused together, the renal substance measuring here 4·3 cm. vertically by 1·5. cm. antero-posteriorly. While the posterior surface is smooth, the organs are markedly lobulated anteriorly and the arrangement of the

lobules suggests a deficiency of the lower part of the right kidney at the point of junction with its larger fellow. Both kidneys possess a reniform outline save that the convex outer margin is somewhat depressed about its middle. In both the hilum is situated anteriorly, and the lobulated renal tissue projects inwards behind it towards the middle line.

The *right pelvis*, which is small, is composed of five separate dilated calices, of which four are situated anteriorly, and lie in vertical series; the fifth is posterior, is situated at the lower and outer part of the hilum, and is not seen in the figure (Fig. 8). At the junction of the calices to form the pelvis (*i.e.* at the lower and outer part of the hilum) is situated a calculus which forms a mould of the pelvis. The calculus is elongated, and at its lower end presents a blunt, conical extremity which lies in the funnel-shaped commencement of the ureter. On its posterior surface a process, 1 cm. long, passes upwards to enter the first (uppermost) calyx, while the upper end of the stone bifurcates into fang-like processes which lie in calices Nos. 2 and 3 of the vertical series. The length of the calculus, including the processes at its upper end, is 3.1 cm., and its greatest thickness is 1.25 cm. transversely and 1.5 cm. antero-posteriorly. It is heavy, hard and glazed and of a dark brown colour, save at the extremities of the upper processes, which are white and apparently phosphatic. The dark brown outer layer seems to have been deposited on a calculus, originally white, and is probably uric acid. The calices and pelvis have been slit up to show the stone.

The *ureter* is apparently normal and lies in a groove between the lower two lobules of the kidney. The *pelvis* projects for a distance of 1.06 cm. beyond the depression in the outer margin of the kidney.

The *left pelvis* is very much dilated and measures 6.25 cm. vertically, by 5 cm. transversely, by 4.3 cm. antero-posteriorly. It is formed by the junction of six calices, four anterior (in vertical series), and two posterior, which latter emerge from the outer part of the hilum. These calices are all very markedly dilated. Viewed from behind, the pelvis projects beyond the outer margin of the kidney for a distance of 3.1 cm.

The *ureter* is much narrowed at its origin, but subsequently increases till its full size is reached at a point 3·75 cm. from the pelvis. It lies in a groove on the anterior surface of the lower end of the kidney.

The *vessels* have not been preserved. Two arteries are seen entering the posterior surface of the fused tissue with a vertical interval of 1·25 cm. between them.

The adrenals were adherent to the kidneys and invaded them. The renal substance was normal otherwise.

The specimen, which was sent by Dr. Primrose, of Belvidere Fever Hospital, was obtained at the necropsy of a male, aged 16 years, the subject of enteric fever. He died on the 22nd day of the disease, after a fortnight's residence in hospital. Albuminuria was noted on the day after admission. There was no note in the history pointing to calculus.

(Pathological Museum, University College, Dundee.)

CASE XI.

Congenital hydronephrosis from narrowing of the ureter at its origin.

The preparation was removed by operation, and consists of a large thin-walled cyst, measuring vertically 13 cm. and transversely 12 cm., on one aspect of which the remains of kidney tissue is flattened out. The cyst is divisible into two portions—pelvic and renal. The former consists of a thin-walled portion much distended, measuring 9 by 7 cm.; the latter is considerably more elongated, measuring 13 cm. from above downwards and 5 cm. transversely. The dilated calices are partially displayed in this latter portion. Appended to the pelvis is a portion of the ureter, which, at its point of section, 5 cm. from the pelvis, is normal in appearance. On approaching the pelvis it tapers, and at its actual entrance measures about 1 mm. It contained colourless, odourless fluid; and on squeezing the contents fluid was observed to drop very slowly from the cut end of the ureter. The internal orifice of the ureter is represented by a slight dimple, and passage is given to a bristle.

The specimen was obtained from a girl, aged six years, who

had a large tumour in the right renal region, which had been first noticed eighteen months after birth. It was stated that it had remained of same size till the date of operation. It was removed by abdominal nephrectomy by Dr. Dalziel. (Western Infirmary *Path. Reports*, No. 5168.)

CASE XII.

Hydronephrosis; great narrowing of pelvic orifice of the ureter.

The kidney, which is the left, presents a marked dilatation of the pelvis and calices, and the latter communicate with the pelvis by comparatively small apertures, expanding inside the kidney so as to form considerable cavities. The ureter is extremely narrowed at its pelvic orifice, so that even a fine probe could not be passed, only a bristle being admitted. No cause for the stenosis was discoverable, and a congenital origin seems the most probable. The specimen was from a male, aged 50, who died in consequence of cerebral softening. (Western Infirmary *Path. Reports*, No. 2305.)

CASE XIII.

Unilateral hydronephrosis with obliteration of the ureter in a congenital cystic kidney. (Figs. 9, 10.)

The specimen was examined and figured after hardening in spirit.

The *right kidney* is of normal appearance, and measures 3.12 cm. vertically by 1.8 cm. transversely. Foetal lobulation is well marked. Its pelvis and ureter also present normal appearances.

The *left kidney* is larger, measuring 4.3 cm. vertically by 1.5 cm. transversely. On the anterior aspect renal substance is present internally and superiorly, while the extremities and the external border of the organ are formed by numerous cysts. Posteriorly the mass is represented entirely by cysts. The *pelvis* is very much distended, and projects inwards for a distance of 1.5 cm. It measures vertically, in the hilum, 2.5 cm. The *ureter* is entirely obliterated at its junction with

the pelvis, being here represented by a fibrous thread of 2·7 mm. in length.

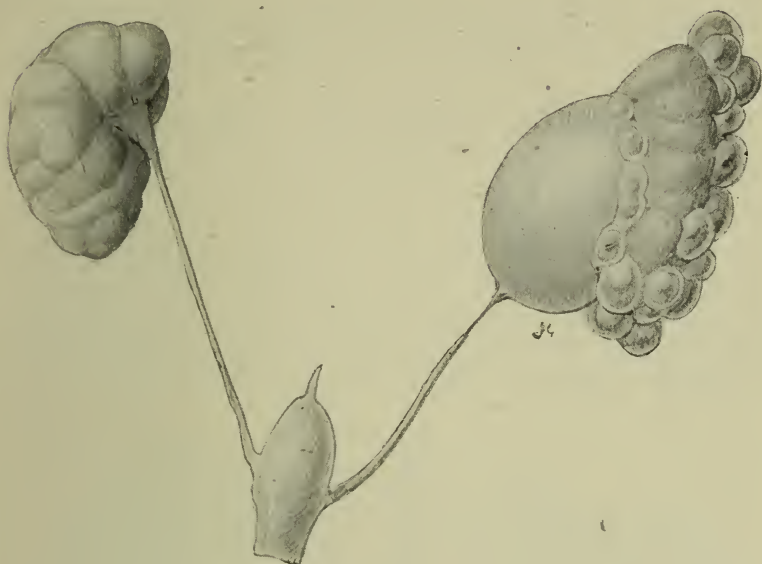


FIG. 9.

On median vertical section (see Fig. 10) the kidney is found to be in a hydronephrotic state. Its substance is hollowed out by two large cavities continuous with the pelvis and extending to within 2·7 mm. of the cortex.

The cysts, already mentioned, are seen to be situated peripherally, and are without connection with the hydronephrotic cavities.

The pelvis was distended with clear watery fluid.

The vascular relations have not been preserved.

Microscopic examination.—Right kidney presents a normal structure. The left shows very marked fibrous

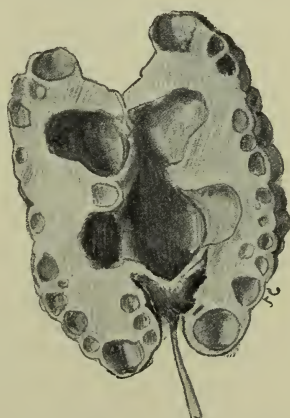


FIG. 10.—Left Kidney split and opened up to show cysts and dilated pelvis.

tissue development, mainly in its pyramidal portions. The glomeruli are crowded together in large numbers in the cortical parts. The cysts are lined by flattened epithelium.

We are indebted to Dr. James Rogers, Assistant Surgeon, Royal Infirmary, Dundee, for the opportunity of examining this case.

The specimen was obtained from a female foetus born between the seventh and eighth months. The presentation was of the breech, and delivery was effected with considerable difficulty. The genito-urinary organs, with the exceptions mentioned, were well formed. A number of other malformations were present, viz. : hydrocephalus, diaphragmatic hernia (containing spleen, stomach, pancreas, and small intestine), branchial cyst, hammer-headed Meckel's diverticulum, etc., etc. (Pathological Museum, University College, Dundee.)

CASE XIV.

Hydronephrosis from branch of artery crossing ureter.

The pelvis of the kidney has been filled with paraffin. It is seen to be enormously dilated, bulging out from the hilum in a conical or pear-shaped form, the hilum being greatly enlarged so as to accommodate the dilated pelvis. There is also a lesser degree of dilatation of the calices. The preparation shows the organ supplied by two principal arteries. The larger of these has four branches, three of which pass into the kidney, along the anterior border of the renal pelvis, whilst the fourth passes rather downwards and backwards. In its passage the artery has crossed the ureter close to its insertion into the apex of the pelvis. The artery here is partly hidden in a deep groove produced by it in the dilated pelvis and the ureter emerges from beneath the artery in this groove. The groove is overhung on the opposite side from the ureter by two rounded bulgings which consist of the apex of the pelvis and the dilated first part of the ureter, the former being thin and translucent, and the latter thicker and opaque from the presence of the muscular coat in the ureter. The ureter could be drawn out from beneath the artery and the

place of crossing was marked by a thinning due to atrophy of the muscular coat.

The specimen was obtained from a male, aged 65 years, who died from bronchitis. There were no known symptoms referable to the kidney. (*Western Infirmary Path. Reports*, No. 2560; also *Glasgow Medical Journal*, vol. xxxv., p. 342; and *Glasgow Pathological and Clinical Society's Transactions*, vol. iii., p. 278.)

CASE XV.

Unilateral hydronephrosis; vessel kinking ureter. (Fig. 11.)

The *right kidney* presents nothing striking on examination. It measures 13.5 cm. vertically by nearly 8 cm. transversely at the upper and 5 cm. at the lower end.

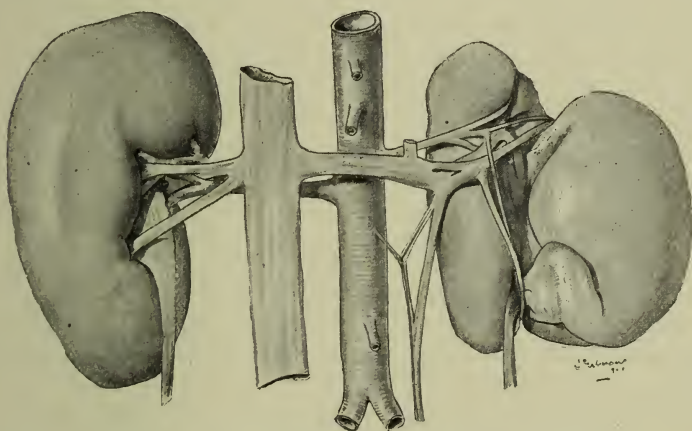


FIG. 11.—The left kidney has been rotated outwards to show the posterior surface of the dilated pelvis.

The *left kidney* is much smaller, measuring 10 cm. in length by 4 cm. transversely at the upper and 2 cm. at the lower end. The organ is irregularly lobular (sacculated) and is somewhat hidden by the enormously dilated *pelvis*, which measures 9 cm. vertically by 5.5 cm. transversely by 4 cm. antero-posteriorly. The dilatation extends into the kidney proper so that the renal margin is represented by a thinned rind of tissue over the attached portion of the pelvis, while the convexity is transparent in

parts. The *hilum* is marked as a cavity posteriorly but not anteriorly. The *ureter* is seen descending from behind the lower end of the dilated pelvis. On viewing the specimen from behind, the dilated condition of the pelvis is explained anatomically by the relation of the renal vessels which seem to embrace the sac. The lower renal vessel has kinked the ureter, causing dilatation of its proximal portion.

Vessels.—The *right renal artery* bifurcates about 3·5 cm. from its origin from the aorta, and 2·5 cm. from the hilum into two divisions. The upper or anterior of these supplies only the upper part of the kidney by means of three branches. Two of these enter the hilum in front of the pelvis while the third enters the kidney directly on its inner surface, well above the hilum. The lower or posterior division breaks up into three branches, which enter the hilum in vertical series, behind the pelvis. The *left renal artery* is not more than half the calibre of the vessel on the right side. It arises from the left side of the aorta at a level slightly higher than its fellow, and after a course of 4 cm. it divides into three branches. Of these the largest supplies the hilum in its upper two-thirds, a smaller twig passing to the convexity of the inner surface of the upper end of the kidney. The remaining two branches embrace the pelvis above and below and the latter vessel kinks the ureter. As a result of the distension of the pelvis the anterior wall bulges through the vascular circle and so the embracing vessels can only be seen on viewing the specimen from behind, while their terminal portions are found in the anterior part of the hilum.

Veins; right.—A large vein passes upwards to the vena cava from the lower part of the hilum, across the front of the pelvis. It joins the vena cava in front of and below the termination of the renal vein proper. This latter receives a large branch from the lower part of the hilum behind, its other tributaries being from the upper part of the hilum and corresponding to the branches of the artery in front of and behind the pelvis.

Left.—A large vein drains the hilum above and behind and companion vessels are found with the arteries embracing the pelvis. These latter join together, separate, and join again, forming a small circle, the lower part of which receives the left

spermatic vein while the proximal part joins with the first-mentioned to form the left renal vein. This joins the vena cava about 2 cm. above the upper right vein.

History was unfortunately not obtained.

CASE XVI.

Bilateral hydronephrosis from kinking of the ureters at their origin, in a child aged 13 months. (Fig. 12.)

The *right kidney* (see figure), which is preserved entire, measures 10 cm. in length by 3.75 cm. transversely at the extremities and 3.12 cm. at its middle. The surface of the organ is fairly smooth, both behind and in front, but in the latter situation a few shallow furrows are present, running transversely to the long axis. The renal tissue is not very thick and is markedly thin anteriorly at the junction of the middle and lower thirds of the organ.

The *pelvis* is so greatly dilated as to form a very prominent feature of the specimen. It measures vertically, in the hilum, 7.5 cm. and it passes inwards towards the middle line of the abdomen for a distance of 5 to 6 cm. It tapers inferiorly to the commencement of the *ureter*, which duct is greatly narrowed in the upper part of its course for a distance of 2.5 cm. In the preserved specimen this narrow portion measures 3 mm. across. When the specimen was received by us, the pelvis contained a quantity of watery urinous fluid, which could be expelled through the ureter on squeezing the pelvic sac.

The *left kidney* is smaller and of firmer consistence. It measures vertically 9.3 cm., and transversely 4.3 cm. at the upper and 1.8 cm. at the lower end of the hilum.

The anterior surface is cleft by deep fissures running in various directions and separating the organ into distinct lobules; the posterior surface is smooth. The *hilum* measures 4.3 cm. vertically, and is situated in its normal position internally, as in the right kidney. The *pelvis* has been somewhat cut up in the removal of the specimen from the body, but it presents the appearances of dilatation, and passes inwards

towards the middle line of the abdomen for about 2.5 cm. The *ureter* has not been preserved.

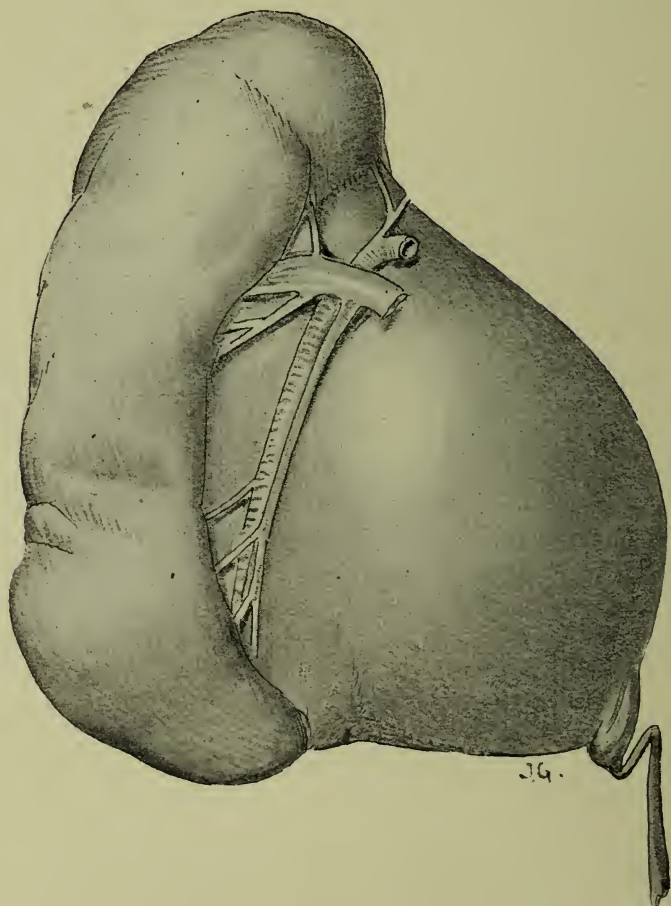


FIG. 12.

This kidney has been split longitudinally and shows, internally, well-marked though not extreme hydronephrotic changes.

In both, the terminations of the vessels have been preserved. These are situated normally, in front of their respective pelves and have caused no obstruction to pelvis or ureter.

The specimens were shown last year to the Southern Medical

Society of Glasgow, by Dr. James Weir, and he has kindly furnished us with notes of the case.

The following is a summary of Dr. Weir's notes:

The patient was a male child, aged 13 months, in whom, after an attack of what was said to have been "acute rickets," a tumour was discovered on the right side of the abdomen. This tumour was believed to be renal. The child was restless and fretful and was said to be passing very little urine. Beyond this last there were no special symptoms observed.

At the *post-mortem* examination there was evidence of rickets. Permission was granted to inspect the abdomen only. The right kidney was removed entire. There was marked hydronephrosis and complete blockage of the ureter, due to its being kinked and bound to the renal pelvis by adhesions. On these being separated urine trickled freely through the urethral canal. The left kidney, which was removed, was also found to be hydronephrotic. It was damaged in removal as, there being no suspicion of the condition being present, the pelvis was cut through and the ureter could not subsequently be found on account of the limited incision through the abdominal wall.

Dr. Weir looked on the condition as possibly "acquired" from prolapse of the kidneys due to a wasting of the perirenal fat as a part of the general emaciation. This prolapse he thought had brought about the kinking of the ureters.

We think it quite as likely that the condition might be congenital, and we have therefore included the case.

CASE XVII.

Hydronephrosis, distended ureters, and bladder from a male infant aged 5 days. (Fig. 13.)

The *right kidney* measures 5.2 cm. vertically by 2.7 cm. transversely at its broadest part, just above the hilum.

The *hilum* is situated on the concave inner aspect of the organ, and contains the somewhat dilated and thinned *pelvis*, which projects inwards for a distance of 1 cm. The pelvis is funnel-shaped and tapers downwards and inwards to the commencement of the ureter.

The *left kidney* is shorter and broader than the right, and shows an approach to the discoid shape. It measures 4·7 cm. vertically by 3·4 cm. transversely. The *hilum* is situated antero-internally and is occupied by the dilated *pelvis*, which measures 2·4 cm. transversely.



FIG. 13.—The anterior wall of bladder cut away and urethra opened from above.

Both kidneys are lobulated on their anterior surface. This is especially marked in the left, which is deeply fissured particularly at its lower extremity. The left kidney is also lobulated posteriorly while the right is comparatively smooth.

On vertical section both kidneys present distinct hydro-nephrosis. The dilatations are much less marked in the right than in the left, where the renal cortex is reduced to a thickness of 3 mm. They do not coincide entirely with the lobulation of the left organ but are certainly a factor in its production.

The *ureters* are distinctly dilated, the dilatation being irregular, and halfway down in the left there is an actual reduplication of the duct. The dilatation is present in the right to within 7 mm. of the bladder-wall, while in the left it ceases suddenly at a distance of 2·5 cm. from that viscus.

The *bladder* is greatly dilated and measures vertically 9·5 cm. by 7·5 cm. transversely in its greatest breadth. Its wall is but 1 mm. thick at the summit, but lower down it measures 3 mm. A pin-hole depression corresponding to the situation of the urachus is present on the inner surface. The urethral orifices are apparently normal and admit a small bristle.

The *urethra*, laid open from above in its entire length, presents no obstruction or narrowing of its lumen. There was no phimesis.

The *testicles* and *vasa deferentia* are of normal appearance. The specimen was obtained *post-mortem* from an infant aged 5 days, who was also the subject of atresia recti (Royal Hospital for Sick Children, *Pathological Journal*, III., No. 104 (374)).

CASE XVIII.

Enormous distension of bladder with diverticula; distension of ureters. Impervious urethra.

The *right kidney* measures 3·75 cm. in length by 1·25 cm. in breadth; the *left* measures 1·8 cm. by 9 mm. Neither kidney on median section shows any naked-eye appearance of hydronephrosis.

Both *pelves* are moderately dilated and their walls are thickened. Both *ureters* are much distended and thickened, especially about their middle part, and in both, but especially in the left, there is a narrowing about 2·5 cm. from the pelvis. The orifice of the right ureter admits the point of the index finger; the orifice of the left, equally large, opens into a diverticulum of the bladder, which admits the terminal inter-node of the thumb. This diverticulum measures 2·5 cm. vertically by 1·25 cm. antero-posteriorly.

The *bladder* is greatly distended, measuring 8·7 cm. vertically by 7·5 transversely. The summit presents, immedi-

ately posterior to the remains of the urachus, a diverticulum of elongated form with its long axis vertical, and measuring 3.1 cm. Its orifice has a diameter of 2.5 cm. The muscular

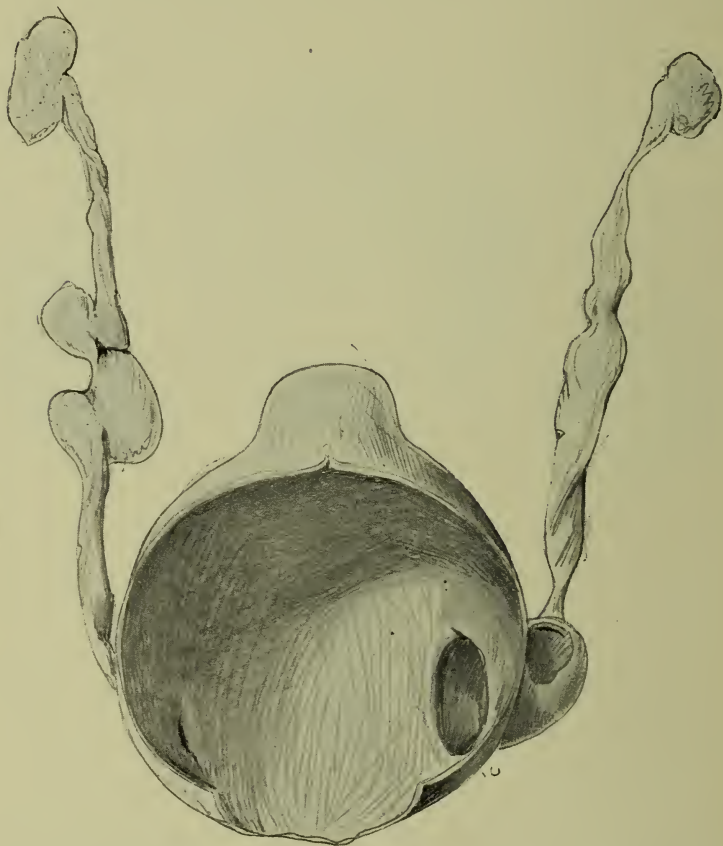


FIG. 14.—The diverticulum at the orifice of the left ureter has been cut open to show the ureter opening in its upper wall.

coat of the bladder forms a well-defined margin round the orifice of this diverticulum, which seems to be formed mainly by a pouch of mucous membrane, with a few muscular fibres spread over it.

Microscopic examination of the kidneys shows in great part replacement of the renal substance by fibrous tissue.

We are indebted to Dr. Halley, Assistant Surgeon, Royal Infirmary, Dundee, for this case. The specimen was obtained from a male infant, the subject of atresia recti and impervious urethra. There was no indication of an anus, and the urethra was found occluded about half-an-inch from the bladder. There was considerable abdominal distension. The child lived for 28 hours. (Pathological Museum, University College, Dundee.)

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